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GOITRE STUDIES: II. RELATIONSHIP OF ENDEMIC GOITRE TO THE FOOD CONSUMPTION PATTERN OF FAMILIES.[1]

By ANITA OSMOND and F. W. CLEMENTS,
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FROM time to time attention has been drawn to the possible relation between diet and socio-economic factors on the one hand and the incidence of endemic goitre on the other. Turton[1] observed in Wirksworth, Derbyshire, that the incidence of goitre at the free church school was 15% compared with 3% among children who attended the council school. The latter group were drawn from a more prosperous type of home. A similar observation was made in Canberra by Clements,[2] who found 65% of endemic goitre among girls aged twelve to fourteen years at Telopea Park Central School, compared with 43% at Canberra High School. In general the intellectual ability and the standard of personal hygiene were higher among the children in attendance at the high school. Olesen and Taylor[3] noted that the nutrition of children with normal glands was "consistently and considerably better" than the nutrition of children with goitre. Mustard and Waring[4] observed the same tendency.

McClendon and Hathaway[5] considered that the consumption of liberal quantities of milk products, leafy vegetables and fruit would act as protection from endemic goitre. This hypothesis was based mainly on theoretical considerations, and was not confirmed by experimental work or by survey. The protection was believed to be due to the high iodine content of these foodstuffs, even if they were produced in an endemic region.

It has been shown[5][6] that foods grown in goitre-free areas contain more iodine than foods grown in goitrous areas. Moreover, the addition of potassium iodide or certain manures to the soil has the effect of increasing the iodine content of the foods grown thereon.[6][7] A comprehensive survey by Hercus and Roberts[6] of the iodine content of foodstuffs raised in goitrous and non-goitrous districts of New Zealand revealed that the foods which contain most iodine are, in order of merit, fish, green vegetables, wholemeal products, milk, meat and root vegetables. In a normal, well-balanced diet (the consumption of large quantities of fish excluded) the greatest contribution to the iodine intake is made by milk, followed by vegetables. Nutritional surveys[8][9] and metabolism experiments[6] have confirmed the observation that persons in different regions who presumably eat the same type of food have a different iodine intake. However, it has not been demonstrated in endemic areas that sufficient iodine can be obtained from dietary sources, fish excluded, to protect all age groups from goitre. The beneficial effects of fish were reported by Lunde.[10]

Another aspect of the relation between diet and endemic goitre is the possible interaction of iodine with other nutrients. This is not a new concept. McCarrison, in 1920,[11] reported an increase in the thyreoid gland weights of scorbutic guinea-pigs. The increase was of the magnitude of 200% to 300%. Sure[12] reported hypertrophy of rat thyreoids accompanying thiamine deficiency, with an increase in the ratio of thyreoid weight to body weight. A number of workers have observed histological changes in the thyreoid gland during acute and chronic scurvy.[13][14] Abercrombie[15] found that these changes are offset by the administration of potassium iodide or thyreoid substance. There is no evidence that the human thyreoid gland is affected in the same manner as the thyreoid of the laboratory experimental animal. However, a number of investigators have observed that thyreoid enlargement in humans is accompanied, in a high per-

[1] The following students of the 1946 School of Nutrition, Australian Institute of Anatomy, Canberra, participated in the survey, visiting the households and assisting the housewives in the recording of the information required: Miss P. Ashworth, Miss M. Braithwaite, Miss E. Cherry, Miss L. Cherry, Miss M. Coleman, Miss D. Langley, Miss M. McArthur, Miss M. Martin and Miss A. Thorburn.

centage of cases, by signs of deficiency diseases. Eason[16] held that 75% of his endemic goitre patients were suffering from chronic anæmia.

This paper is a report of a nutrition-dietary survey which was carried out in the East Gippsland district of Victoria with the purpose of investigating the possible correlation between endemic goitre and diet. The survey was made simultaneously with the clinical survey of thyreoid enlargement which has been reported previously, and with the biochemical survey of urinary iodine excretion.[22][24] The data on food consumption were collected by the log-book or inventory method.

Choice of Area.

An area was required which was (*a*) situated in a goitre belt, (*b*) supplied with milk, green vegetables and, if possible, meat and eggs from one or comparable sources, to ensure that the iodine intake from food was dependent only on the quantities of foods eaten, and (*c*) suitable for the conduct of a food consumption survey.

East Gippsland conformed with these requirements. Data were collected for households in the following localities: Orbost (population 2000), which is situated on the Snowy River flats; Bruthen (550) on the Tambo River Flats, Lindenow, Hillside, Sarsfield, Wy-Yung and Lucknow, which are on the river flats in the vicinity of Bairnsdale, and Swift's Creek (600) and Omeo (500) on the high plains. Bairnsdale and Sale were excluded, as the industrial development which has taken place there has been accompanied by urban influences which make these towns different in character from the remainder of the area.

The high incidence of thyreoid enlargement in East Gippsland is well known[17][18][19][20][21] and was confirmed by the present clinical survey.[2]

The district is practically self-sufficient with respect to perishable foods. Economically the area is dependent on agriculture, pastoral pursuits and the timber industry. By Australian standards the holdings of land are small, the majority being of a size which can be worked by one man for most of the year. The farms are organized to produce as much as possible of the family's requirements of food. This peasant way of life is reflected among breadwinners who follow an urban occupation, many of whom have enough land around their home to run a cow, keep fowls and raise vegetables.

The total supply of milk, meat and eggs is produced within the area. Half of the households in the district own a cow, and only Orbost and Bruthen have a door-to-door delivery of milk. Where supplementary feeding of dairy cattle is necessary, locally grown fodder is used. There is some trade in meat within the district. Butchers purchase their own beasts as livestock and top them off locally before slaughtering them.

Vegetable growing for the city markets is a major industry on the river flats; but the local demand for vegetables is met for only a short period each year. Vendors purchase the bulk of their supplies from the Melbourne markets. This means there are two sources of vegetables, local and imported. Households with vegetable gardens use imported vegetables only as a supplementary supply. Households which do not grow vegetables consume locally grown vegetables for part of the year only.

Food Consumption Survey.

Sampling.

The households from which data were obtained were selected from the children in attendance at the schools in the area chosen. These children were examined for thyreoid enlargement in the course of the clinical survey, and the random selection of boys whose twenty-four hour urinary excretion of iodine was measured was made from the same group.

The cooperation of housewives with the supervisors who assisted them to make the records was good, but for various reasons all the households in the original sample were not able to participate. The most frequent causes of non-cooperation were absence from the district during the survey period and duties outside the home. This possible element of bias is an unavoidable defect of studies of this nature.

The number of households investigated was 112 and the distribution through the area is shown in Table I.

TABLE I.
Distribution of Households Covered by the Survey.

Place.	Number of Households.
Orbost	31
Bruthen	20
Lindenow	11
Hillside	6
Sarsfield	3
Wy-Yung	3
Lucknow	9
Swift's Creek	17
Omeo	12

The occupations of the heads of the 112 households were as follows: rural work, fishing and hunting, 35; timber industry, 27; professional and semi-professional work, 6; administrative, commercial and clerical work, 17; skilled manual work, 9; unskilled manual work, 7; protective services, 3; no occupation, 5; occupation unstated, 3. Occupations have been grouped according to the classification of the Commonwealth Bureau of Census and Statistics.[23]

Altogether 643 persons were covered by the survey. Sixteen households contained eight or more persons, and 32 households contained seven or more persons. Three pregnant women and six nursing mothers were present in the total number (Table II).

Technique.

Every effort was made to follow the survey technique outlined in the report of the survey of the "Food Consumption and Dietary Levels in 2370 Australian Family

TABLE II.
Persons Covered by Survey

Area.	Number of Homes.	Number of Persons.	Number of Adults.					Number of Children.							
			Males (15 to 60 Years).	Females (15 to 60 Years).	Persons (60+ Years).	Pregnant Women.[1]	Lactating Women.[1]	Total Children.	Age 1 to 2 Years.	Age 3 to 4 Years.	Age 5 to 6 Years.	Age 7 to 8 Years	Age 9 to 10 Years.	Age 11 to 12 Years.	Age 13 to 14 Years.
Orbost	31	179	32	41	1	2	1	105	18	9	20	15	21	12	10
Bruthen	20	110	19	27	1	1	—	62	8	10	11	14	11	5	3
Lindenow, Hillside, Sarsfield, Wy-Yung, Lucknow	32	206	44	41	3	—	4	118	18	18	18	14	26	16	8
Swift's Creek, Omeo	29	148	34	32	2	—	1	81	9	9	12	18	14	13	6
Total	112	643	129	141	7	3	6	366	53	46	61	61	72	46	27

[1] Included also in females, fifteen to sixty years.

TABLE III.

Comparison of Mean Daily per Capita Intake of Calories and Nutrients with Mean Daily per Capita Recommended Allowances.

Area.	Calories.	Protein. (Grammes.)	Calcium. (Milligrammes.)	Iron. (Milligrammes.)	Vitamin *A*. (International Units.)	Thiamine. (γ)	Ascorbic Acid. (Milligrammes.)
Orbost—							
Intake[1]	2797	88	867	14·8	3766	1210	84
Recommended allowance[2]	2180	59	842	9·1	3313	917	54
Bruthen—							
Intake	2642	84	731	14·2	3125	1146	85
Recommended allowance	2144	58	822	8·8	3200	885	52
Lindenow, Hillside, Sarsfield, Wy-Yung, Lucknow—							
Intake	2994	93	1020	15·2	4016	1203	92
Recommended allowance	2182	59	828	8·9	3301	915	54
Swift's Creek, Omeo—							
Intake	2799	89	873	14·3	3626	1110	87
Recommended allowance	2253	60	812	9·0	3341	928	54
Gippsland, 1946—							
Intake	2826	89	888	14·7	3687	1170	87
Recommended allowance	2192	59	827	8·9	3296	913	53
Victorian country, 1944—							
Intake	2626	81	747	13·7	4190	1091	94
Recommended allowance	2459	60	820	9·1	3339	1043[3]	53
All Australia, 1944—							
Intake	2597	82	714	13·9	3890	1078	97
Recommended allowance	2234	59	816	9·0	3036	1037[3]	53

[1] Mean daily *per capita* intake.
[2] Mean daily *per capita* recommended allowance.
[3] Calculated from National Research Council Recommended Dietary Allowances, 1943.

Households in 1944".[22] The methods for the collection of data were identical with those employed in 1944, but the collection presented some difficulties which had not been encountered previously. More than half of the homes in the random sample owned a cow, and it was common for housewives to make their own butter and cream. Meticulous care was required to determine the quantity and nature of the milk products which were used in the home and the quantities which were sold, wasted or fed to the farm animals. Similar difficulties were encountered among the households which slaughtered their own meat. The treatment of home-grown vegetables and fruits has been described in the report of the 1944 survey.

The methods for the statistical treatment of the material were identical with those employed in 1944, with one exception. The standard against which Calories and nutrients were assessed in 1944 was the National Research Council, Recommended Dietary Allowances (restricted), 1943.[24] For the present investigation the National Research Council Recommended Dietary Allowances (restricted), Revised, 1945,[25] were employed. In so far as this survey is concerned the revision has affected the recommended allowances for thiamine, which are somewhat lower in the revised scale than those recommended in 1943.

Results.

The mean daily *per capita* intake of Calories and the specific nutrients for the households covered by the present survey and for Victorian country towns and all Australia, according to the 1944 survey, are shown in Table III, together with the recommended allowances, prescribed by the National Research Council of America, calculated on a *per capita* scale.

The mean intake for the 112 households exceeded the mean recommended allowances for Calories and all nutrients. For Bruthen, the mean *per capita* intake of calcium failed to meet the mean *per capita* recommended level of intake by 11%, and the mean *per capita* intake of vitamin *A* also was slightly lower than the recommended allowance.

The variations which occur between households are masked in Table III. The numbers of households in each area which fell below the recommended levels are shown in Table IV.

Diets have been graded into four arbitrary categories for purposes of comparison. The categories are based upon the total of the deficiencies of Calories and nutrients for each household. The four categories are as follows: A grade, 10% or less total deficiencies; B grade, 11% to 30% total deficiencies; C grade, 31% to 60% total deficiencies; D grade, 61% or more total deficiencies. The results of this survey, according to the scale listed above, are given in Tables V and VI, together with those obtained for Victorian country towns and all Australia in 1944, when the same arbitrary classification was used.

Discussion of the Results.

The results indicate that the intakes of Calories, protein, iron, thiamine and ascorbic acid were satisfactory for nearly all households, and that the intakes of calcium and vitamin *A* were unsatisfactory, compared with the recommended level of intake, for a large percentage of households.

Forty-six *per centum* of households failed to attain the recommended level of intake for calcium and the average percentage deficiency was 21·3 (Table VII). Although this is a more satisfactory position than was found in the Victorian country towns in 1944, it is an unexpectedly large number when it is considered that 59 households, or 53% of the total number, owned a cow in milk. Of the

TABLE IV.

Number of Households Falling Below Recommended Allowances for Calories and Nutrients.

Area.	Number of Households.	Calories.	Proteins.	Calcium.	Iron.	Vitamin *A*.	Thiamine.	Ascorbic Acid.
Orbost	31	3	—	16	—	14	(5)[1] 1	3
Bruthen	20	2	—	14	—	11	(5) 3	1
Lindenow, Hillside, Sarsfield, Wy-Yung, Lucknow	32	1	—	11	1	10	(5) 2	1
Swift's Creek, Omeo ..	29	3	—	10	1	14	(12) 4	3
Total	112	9	—	51	2	49	(27) 10	8

[1] Figures in parentheses are the numbers of households which failed to meet the Recommended Dietary Allowances, 1943.

53 households which did not own a cow, 36, or 67·9%, failed to meet the recommended allowance according to the composition of the household, compared with 14, or 23·7%, of the 59 households in possession of a cow. Table VII shows also that the average percentage deficiency was lower for the latter group. It is obvious, therefore, that the necessity to purchase milk is an important factor influencing the calcium intake.

The reasons for the low calcium intake among households in possession of a cow were as follows: (i) the reservation of a large part of the milk yield for cream and butter; (ii) the practice of feeding the skimmed milk to farm animals; (iii) the wastage of skimmed milk; (iv) the use of cream as a substitute; and (v) a dislike for milk.

TABLE V.
Numbers of Households Falling into Each Arbitrary Diet Grade.

Area.	A.	B.	C.	D.
Orbost	15	7	3	6
Bruthen	7	6	4	3
Lindenow, Hiliside, Sarsfield, Wy-Yung, Lucknow	20	7	2	3
Swift's Creek, Omeo	16	4	4	5
Total	58	24	13	17

TABLE VI.
Percentages of Households in Each Grade, Compared with Victorian Country Towns and All Australia, 1944.

Area.	A.	B.	C.	D.
Gippsland, 1946	52	21 (20)[1]	12	15 (16)[1]
Victorian country, 1944	40	20	19	21
"All Australia", 1944	32	21	21	26

[1] Figures in parentheses were obtained when thiamine intake was assessed against the 1943 recommended allowances.

It will be noted that 49 households failed to meet the recommended allowances for vitamin *A*, according to the composition of the household. This is surprisingly high, as 59 households had a cow in milk at the time of the survey, and thus the consumption of butter was not restricted to the ration and cream was available. The explanation is not to be found in the dietary pattern of the households, but in the apparently unjustifiably high figures set for the recommended allowances for vitamin *A*. Nutrition authorities in different parts of the world are aware of this, and attempts are being made to find a more satisfactory set of figures.[26]

The data for thiamine (Table IV) reveals that a substantial percentage of households had a thiamine intake which was unsatisfactory if the recommended allowances, which were proposed by the Food and Nutrition Board of the National Research Council in 1943, are taken as a standard, but satisfactory if the recommended allowances revised in 1945 are taken as a standard. The 1944 Australian survey, for which the 1943 figures were employed, showed that 44% of households in Victorian country towns and 20% to 66% of households in other areas fell below the recommended allowances for thiamine. It is probable that if the 1945 allowances were used as a basis for comparison, these percentages would be reduced.

Superficially, the intake of ascorbic acid appears to be satisfactory. In the field, however, the investigators gained the impression that the ascorbic acid intake was low. Little citrus fruit is eaten, as most of the supply is imported into the district and is highly priced. The mean *per capita* intake of ascorbic acid from fruit, tomatoes and fruit products was 23 milligrammes per day. The mean *per capita* recommended level of intake was 54 milligrammes per day. It is likely that a deficiency of this nutrient is one of the defects of the Gippsland dietary.

Relationship between Household Diet and the Existence of Endemic Goitre in Children of the Household.

Households were classified into three categories, goitrous, non-goitrous and mixed, according to the clinical findings.[2] When the older children in the family all had a goitre, the classification was "goitrous" irrespective of the condition of the thyreoid gland in the younger children. A "mixed" household was one in which one child or more was goitre-free while a younger child had an enlarged thyreoid. If an older child was goitrous and a younger child was goitre-free, the grading was made according to the age of the younger child. Families of which less than two children were examined for thyreoid enlargement and families which had resided in the district for less than two years were excluded.

TABLE VII.
Mean Percentage by which Households which did Not Meet the Recommended Allowance for Calcium Fell Below the Allowance.

Area.	Households Falling Below Recommended Allowance.		Mean Deficiency. (Percentage.)
	Number.	Percentage.	
Gippsland	51	46	21·3
Gippsland, 1946; households owning a cow	14	24	16·7
Gippsland, 1946; households without a cow	36	68	23·5
Victorian country towns, 1944	267	61	27·4

Table VIII shows that there was no relation between the diet grading of the households and the incidence of endemic goitre among the children. Deficiencies of the specific nutrients and the percentages of the recommended allowances for ascorbic acid provided by fruits were compared with the classification of households for goitre, and in no case was a relationship established. There was no difference in the incidence of thyreoid enlargement among children from families which grew their own vegetables and families which purchased supplies.

The significance of this finding must be considered against the background of the ætiology of endemic goitre. This condition is the result of a number of factors, including diet, which have been in operation over a period of years. A food consumption survey gives the apparent food pattern of the households or its individual members at the time of the survey, and this may not represent the conditions that prevailed during the time of the development of the goitre. In view of this, the absence of a correlation is not unexpected.

TABLE VIII.
Comparison of Diet Grading with the Incidence of Goitre Among School-Children.

Classification of Households.	Grade A.		Grade B.		Grades C and D.	
	Number.	Percentage.	Number.	Percentage.	Number.	Percentage.
Goitrous (24)	12	50	5	21	7	29
Non-goitrous (24)	12	50	7	29	5	21
Mixed (27)	13	49	7	30	7	21

Relationship between Diet and Urinary Excretion of Iodine.

Nineteen of the 36 boys whose twenty-four hour urinary excretion of iodine was measured[27] were members of a household for which a food consumption record was collected. Table IX shows that the diet grading did not bear a relationship to the urinary excretion of iodine. The numbers are too small for any definite conclusions to be drawn.

TABLE IX.

Comparison of Diet Grading with the Twenty-four-Hour Urinary Excretion of Iodine.

A Diets. (γ)	B Diets. (γ)	C Diets. (γ)	D Diets. (γ)
9·4	22·2	5·8	9·1
26·3	4·1	12·7	19·7
8·0	1·2	8·5	13·3
12·1	3·5		4·3
5·2			5·2
21·5			20·6
Mean 13·8	7·8	9·0	12·0
Mean of A and B 11·4			
Mean of C and D 11·0			

Summary.

1. A food consumption survey by the weekly inventory method was made for 112 households in the East Gippsland district of Victoria.

2. No relationship was found between the diet of the household and the existence of endemic goitre in the children.

3. No relationship was found between the diet of the household and the urinary excretion of iodine of school boys.

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ISO-AGGLUTININS IN CORD BLOOD.

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INVESTIGATIONS on the relationship of Rh antibodies in the maternal and infantile blood have once again drawn our attention to the question of the relationship of anti-A and anti-B iso-agglutinins in the maternal and infantile circulation.

The development of the blood groups in the fœtus has been studied by von Dungern and Hirszfeld, by Schiff, by Kemp and by others, who have shown that the agglutinogens of the red cells are already demonstrable as early as the third month of intrauterine life.[1] Oluf Thomsen and Karsten Kettel, in an extensive series of investigations, have found that the strength of the A and B antigens of the red cells at birth is approximately one-fifth to one-seventh of that of fully developed red cells. The sensitivity increases gradually and reaches its full strength at puberty, remaining unaltered from then onwards.[2] The fœtus therefore develops its blood group antigens very early, and after having provided her share of necessary genes, according to the Mendelian law, the maternal host has no further influence on them.

The origin and development of the iso-agglutinins in late pre-natal and early post-natal life, however, are not so clearly defined. According to Kirihara,[3] Hirszfeld and Zborowski[4] and others, the fœtus does not form its own iso-agglutinins, and any anti-A and anti-B agglutinins found in the cord blood are of maternal origin. Morville, as quoted by Thomsen and Kettel,[1] states that agglutinin production does not start before the third to the sixth month of post-natal life. Thomsen and Kettel found very low agglutinin titres between six and twelve months, and a relatively rapid rise in the first two years, to a peak reached as early as the fifth to the tenth year. From then onwards the titre gradually decreased, and in the age group between ninety and one hundred years the levels were as low as those found in the first twelve months of life.

Liedberg,[5] on the other hand, has occasionally encountered agglutinins in the cord blood which were absent from the mother's serum, and therefore he presumes that the fœtus is sometimes capable of forming its own antibodies.

Further observations on this subject, based on the investigation of 355 samples of cord blood and corresponding maternal blood, are recorded in this paper.

Technique.

Blood group determinations were carried out on tiles, potent typing sera being used. The use of these is

particularly important when the blood of the newborn is being tested, as the agglutinogens are weak and the results cannot be confirmed by the pattern of the agglutinins in the serum. The investigations on infants were carried out on cord blood. Serological tests on the maternal blood were carried out on samples obtained shortly after delivery.

The blood used was collected into dry tubes without any anticoagulant. Sufficient red cells for identification of agglutinogens could be removed from the clot and suspended in normal saline solution to give a 2% to 3% cell suspension. Wharton's jelly only rarely interfered with the determination of blood groups. In a few doubtful cases fresh samples of capillary blood were collected from the heels of the infants. Tests for the presence of agglutinins and of group-specific substances were carried out on serum obtained from the cord blood samples. In order to detect iso-agglutinins samples of the serum were mixed with A, B and O cells and the mixtures were centrifuged for approximately one minute. If agglutinins were found, their strength was titrated on tiles. This method is less sensitive than the centrifugation method, but in this particular investigation we were not concerned with the actual end-point of the titre, once the presence of antibodies was established, but with a comparison of maternal and infantile titres, for which it was adequate. The presence or absence of group-specific substances in the cord serum was tested by mixing it with pooled group O serum of moderately high titre, or with the maternal serum if it was of appropriate group. Group O serum was particularly suitable, as it made it possible, in cases in which the child belonged to group A or B, to differentiate between specific and non-specific absorption. In some cases the results were compared with those obtained by absorption with saliva.

Results.

The distribution of blood groups in mothers and children was as shown in Table I.

TABLE I.

Mothers.[1]		Children.[2]	
Group O	173 (48·7%)	Group O	161 (45·3%)
Group A	127 (35·8%)	Group A	131 (36·9%)
Group B	44 (12·4%)	Group B	54 (15·2%)
Group AB	11 (3·1%)	Group AB	9 (2·5%)

[1] Classified according to red cell antigen and agglutinin content in serum.
[2] Classified according to red cell antigen.

The percentages in the maternal blood groups correspond to the figures obtained and reported earlier on a much bigger cross-section of the population.[6] There are possibly more group B babies than would be expected in a sample of 355, but the distribution of the remaining three groups is within the limits of expectation. It is probable that some of the babies classified as of group B are of group A_2B, with a very weak A_2 antigen which failed to react with anti-A agglutinin.

The results of the observations obtained on this series of maternal and cord blood samples can be tabulated as shown in Table II.

The results obtained reveal four instances in which the agglutinins found in the cord blood were antagonistic to the child's red cells and therefore were presumably of maternal origin (see mother-child relationship 7 and 11 in Table II), and one instance in which they must be of infantile origin (see mother-child relationship 14 in Table II). In the remainder the agglutinins could be of either maternal or infantile origin; the statistical analysis of these cases is given in Table III.

Discussion.

The critical evaluation of these results can be discussed from two aspects.

On the one hand we may consider the child as a serologically independent individual, with a well-defined blood group developed according to the Mendelian law and in accordance with Landsteiner's rules. However, the influence of the maternal-infantile relationship must also be taken into account. We know that there really is no blood independence during intrauterine life. Maternal and infantile circulations are not strictly separated, and any appraisal of results of investigations on cord blood should be partly based on a recognition of this process of interchange. Bearing this in mind, we would expect a filtration of maternal antibodies into the infant's circulation. Such passive filtration has been shown in many cases in respect of acquired antibodies, a fairly high level of which directly related to that found in the maternal blood may be demonstrable in the child's circulation. These may be of bacterial

TABLE II.

	Blood Group Relationship of Mother and Child.		Agglutinin Content of 355 Samples of Cord Blood.				Total.
			No Agglutinin.	Anti-A Agglutinin Only.	Anti-B Agglutinin Only.	Anti-A and Anti-B Agglutinin.	
1	O	O	16	22	8	65	111
2	A	A	57		20		77
3	B	B	15	11			26
4	AB	AB	4				4
Total number in which mother and child were of the same group							218
5	A	O	29		13		42
6	A	B	6				6
7	A	AB	2	1			3
8	B	O	5	5			10
9	B	A	7				7
10	B	AB	1				1
11	O	A	18	1	24	2	45
12	O	B	3	14			17
13	AB	A	2				2
14	AB	B	3	1			4
Total number in which mother and child were of different groups							137

origin—for example, diphtheria, tetanus or staphylococcal antitoxins, or antibodies against viruses, such as those causing herpes, influenza or mumps. Similarly, Rh antibodies, which can be regarded as acquired blood factor antibodies, can be filtered into the child's blood and can be demonstrated if sensitive methods are used. If the child is Rh-positive, their amount is usually small, as the infantile red cells in such cases absorb them rapidly. But two instances have been encountered in which the babies were Rh-negative; in these the maternal and infantile serum had the same concentration of Rh-antibodies (the titre being eight in one case and 120 in the other), the Rh antibodies in the maternal blood having been formed during previous pregnancies.

The serological findings in respect of anti-A and anti-B agglutinins in cord blood, however, are quite different. Even in homospecific pregnancies, in which any absorption of maternal antibodies by the infant's A or B antigen can be excluded, agglutinins corresponding to the maternal group are often missing.

For this phenomenon different explanations are possible.

1. It could be postulated that the infantile part of the placenta, the amniotic fluid and the organs of the fœtus absorb the incompatible antibody—a process which would start relatively early, as group-specific substances in the fœtal organs are already present after the sixth month of gestation. But this hypothesis would explain the selective absence of antibodies antagonistic to the child's agglutinogens only in heterospecific pregnancies, and when mother and child belong to the same group we should expect the cord serum to contain agglutinins. However, the complete qualitative maternal antibody pattern has been found only in 96 samples of cord blood in 214 cases of homospecific pregnancy. (In this paper the term homospecific is restricted to those cases in which the A B O group of mother and child was the same.)

TABLE III.

Category.	Blood Group Relationship of Mother and Child.		Agglutinins in Cord Blood.						χ^2.	*P*	Interpretations of Value of *P*.
			Anti-A Agglutinin.			Anti-B Agglutinin.					
			Absent.	Present.	Total.	Absent.	Present.	Total.			
1	O B	O B	24 15	87 11	111 26				13	0·002	Highly significant.
2	O O	O B	24 3	87 14	111 17					0·5	Not significant.
3	B B	O B	5 15	5 11	10 26					0·47	Not significant.
4	O B	O O	24 5	87 5	111 10					0·058	Nearly significant.
5	O B	B B	3 15	14 11	17 26					0·01	Significant.
6	O A	O A				38 57	73 20	111 77	29	0·001	Highly significant.
7	O O	O A				38 19	73 26	111 45		0·2	Not significant.
8	A A	O A				29 57	13 20	42 77		0·6	Not significant.
9	O A	O O				38 29	73 13	111 42		0·0001	Highly significant.
10	O A	A A				19 57	26 20	45 77		0·0005	Highly significant.

2. The possibility that a high titre of maternal antibodies would lead to an easier overflow of agglutinin into the cord blood was investigated by analysing the results found in children of homospecific pregnancies; thus the problem of secretor and non-secretor states of the infant was avoided. The cases were divided into two groups according to the presence or absence of cord agglutinins, and a comparison was made of the mean titre values of the maternal serum in both groups. The mean maternal anti-A titre (groups O and B mothers) in the group in which agglutinins were present in the corresponding cord blood was 121; in the group in which no agglutinins were present it was 140. The mean anti-B titre (A and O) for the first group was 77, for the second group 55. These differences are not great enough to support the theory of a quantitative filtration. Furthermore, no close correlation could be found between the maternal and infantile agglutinin titres. In one case in which the maternal titre was 2000 no agglutinins were found in the cord blood, and the highest infantile titre (16) was found in a child whose mother's titre was 64.

TABLE IV.

Difference Between the Frequency of Anti-A and Anti-B Agglutinins in Cord Blood of Group O Children of Group O Mothers.

Agglutinin.	Absent.	Present.	Total.	χ^2.	*P*.	Significance.
Anti-A ..	24	87	111	4·97	0·027	Significant.
Anti-B ..	38	73	111			

3. Since it is known that naturally occurring α and β agglutinins are of larger molecular size and have a higher electrophoretic mobility than immune globulins,[7] it is reasonable to assume that the former cannot pass the placental barrier as readily as the latter. Whatever the explanation may be, the figures suggest that the placenta may have some faculty of selective retention for iso-agglutinins, for in the majority of cases no antibodies are encountered in the infant's circulation which would be harmful to its own red cells. However, there is evidence that this selective retention occasionally breaks down; in this series four cases have been encountered in which agglutinins were found in the cord serum which were antagonistic to the child's cells, but which corresponded to the maternal group. The first instance was in the group AB child of a group A mother. The child had anti-B agglutinins of titre four. The child was a secretor; the maternal serum had a titre of 64 at delivery and showed no rise on the eighth day, but at this time it hæmolysed B cells at a dilution of one in four at room temperature. The child was normal at the time of discharge from hospital, and six weeks after birth had a hæmoglobin value of 105% (14·7 grains per 100 millilitres) and a red cell count of 5,800,000 per cubic millimetre. The second child belonged to group A and the mother belonged to group O and had iso-agglutinin titres of 128 (anti-A) and 64 (anti-B). The undiluted cord serum contained anti-A agglutinins. The presence or absence of group specific substances was not investigated and the maternal serum was not tested for a rise in titre during the puerperium. The child developed slight jaundice on the third day, which disappeared on the tenth day. The third case was that of a group A child of a group O mother. The infant, who was a secretor, had anti-A and anti-B agglutinins in the undiluted cord blood. At the third month of pregnancy the maternal serum had titres of 1024 (anti-A) and 64 (anti-B), which were unaltered at delivery. Seven days later they had risen to 8000 (anti-A) and 250 (anti-B). The child was never jaundiced; on the third day its hæmoglobin value was 130% (18·2 grains per 100 millilitres) and its red cell count 5,560,000 per cubic millimetre. In the fourth case the child was of group A and the mother of group O. The child was a secretor and had anti-A and anti-B agglutinins in its undiluted cord serum. The maternal serum had titres of 256 (anti-A) and 64 (anti-B), which did not rise in the first ten days of the puerperium. But on the tenth day the serum hæmolysed A cells in a dilution of one in four at room temperature. The child was two months premature and died within the first twenty-four hours.

These findings, though small in numbers, lead to speculation on the nature of these placenta-permeating antibodies. In three of the four cases (and only these three were fully examined) the children were secretors and as such capable of immunizing the mother. This immunization had apparently taken place and had led in one case to a rise in the iso-agglutinin titre and in two to the development of iso-hæmolysins. The maternal blood, therefore, may contain two kinds of antibodies, the naturally occurring iso-agglutinins, and a second type developed by an immunizing process. Though both react

similarly with red cells, they may be different in structure and size, and only the immune type may be capable of penetrating the placenta, as do Rh or other acquired immune antibodies.

There is also some clinical evidence that the placental blockage of group-foreign antibodies is not complete. In a fairly large series of investigations of newborn babies it was found that the hæmoglobin level of children of heterospecific pregnancies is lower than that of children of homospecific pregnancies.[8] We may, perhaps, assume that this finding is due to the gradual destruction, during intrauterine life, of a small proportion of infantile red cells by maternal antibodies. The antibody-antigen interaction seems to terminate fairly quickly, as the Coombs test, which would indicate the presence of sensitized cells, generally produces a negative result in heterospecific pregnancies.

So far, the agglutinin pattern of the cord blood has been considered in relation to the mother's blood group. An alternative approach is to consider it as an expression of the child's serogenesis. The most striking feature, from this point of view, is the incompleteness of the agglutinin pattern, which varies greatly from that of adult blood. Lack of group-specific agglutinins is hardly ever encountered in serologically fully developed persons. In a large series of routine examinations of the blood of Red Cross donors and of women attending the Queen Victoria Hospital Ante-Natal Clinic, no blood samples were found which did not show the expected agglutinins. In the cord blood, however, lack or incompleteness of isoagglutinins seems to be more the rule than the exception. The agglutinins, if present, are of a very low titre and, apart from the four cases mentioned, are never antagonistic to the child's blood group or, apart from one case, to the maternal group (child group B, mother group AB, anti-A agglutinins present in the cord serum). From these investigations it is not possible to draw any conclusions about the origin of the cord blood agglutinins. If they are of infantile origin and as such express the beginning of a serological ripening process (which is apparently completed as early as the tenth year of life), it is somewhat astonishing that they are missing at birth in approximately 60% of children in our series and in 56·6% in a series of Polayes *et alii*,[9] since a more uniform onset might be expected for a process which is apparently independent of an immunizing stimulus and is regulated only by genetic rules. On this basis the incompleteness of the agglutinin pattern in, for instance, group O children of heterospecific pregnancies, or the complete lack of appropriate agglutinins in the blood of group A and group B children, if their mothers belong to group B and group A respectively, could in heterospecific pregnancies be explained by the assumption that the agglutinins, if formed, were passed back through the placenta into the maternal circulation and absorbed. Since no correlation could be found between secretor and non-secretor state of the mother and the presence or absence of agglutinins in the cord blood, it would seem that the maternal red cells must be the absorbing agent. However, this hypothesis does not explain the lack or incompleteness of infantile agglutinins in those cases in which mother and child both belong to group O.

We have not attempted to follow the development of the agglutinin content of the infantile blood in the first few weeks of life. But Smith,[10] who has studied this aspect, reports that the agglutinins present at birth disappear and that after an interval the full agglutinin pattern corresponding to the child's blood group is developed.

His observation strongly supports an assumption that the cord blood agglutinins are of maternal origin. A few of our observations on twin pregnancies also support this theory. In five cases of homospecific twin pregnancies the agglutinin pattern and agglutinin content as shown by titration were similar in both twins, although in four cases they were not identical twins, as could be shown by the difference of sex or of Rh types. In a sixth case one twin was of group O and the other of group A, but in the serum of neither of them could agglutinins be demonstrated. The statistical analysis of our figures also supports the theory of a maternal derivation of the agglutinins in cord blood. For only in those combinations in which the maternal blood groups are altered are any significant or almost significant differences observed (categories 1, 4, 5, 6, 9 and 10 in Table III), the differences being not significant when the child's blood group is altered (categories 2, 3, 7 and 8).

The analysis also reveals that the appearance of either anti-A or anti-B agglutinins in cord blood is more frequent when the mother is of group O than when she is of group A or group B respectively (categories 1, 4, 5, 6, 9 and 10). The following hypothesis is a possible explanation for these experimental findings. The anti-A and anti-B agglutinins in group O blood are not identical with those in group B and group A blood, although their action on human red cells is similar. Supporting evidence for this assumption for at least anti-A agglutinins is the fact that the agglutinability of A_1 and A_2 cells does not differ so much in group O serum as it does in group B serum (this finding is usually explained by the different distribution of α and α_1 in these two groups), and Kramer's observation on the difference in the thermal amplitudes of anti-A agglutinins. He found that most of the group O sera act on A_1 and A_2 cells almost as strongly at 50° C. as they do at 37° C., but that group B sera, on the other hand, show almost no activity on either A_1 or A_2 cells at 50° C.[11]

A third fact which emerges from the statistical analysis of the figures is that as a whole the anti-A agglutinins are more frequent in cord blood than the anti-B agglutinins (see Table IV). Since in adult group O blood also the anti-A titre is as a rule higher than the anti-B titre, it is posible that both these phenomena could be explained by a greater sensitivity of A cells than B cells. Hirszfeld[12] has a theory, based on agglutination obtained with anti-O serum, that all groups have a certain amount of O antigen in their cells, the amount being in decreasing order in O, A_2, B and A_1 cells. From this scheme it can be postulated that B cells, having more O substance than do A_1 cells, have therefore less B substance than A cells have A substance. On this assumption, B cells are therefore less sensitive in detecting anti-B agglutinins than are A cells in detecting anti-A agglutinins, and the apparent predominance of anti-A agglutinins is due only to a more sensitive testing agent.

The fourth fact which arises from our figures is that the appearance of agglutinins in cord blood, even in the same mother-child combinations, is not regularly related to the maternal titre. The evidence available does not provide a complete explanation of this phenomenon, but Friedenreich and With[13] show that β agglutinin consists of at least three partial agglutinins β_1, β_2 and β_3; Dahr[14] described two partial α agglutinins which he designated as x and y; and Bryce and Jakobowicz[15] postulated a third partial α agglutinin, z. It is possible that these partial agglutinins may in different combinations produce composite agglutinins of varying constitution, and that some of these can pass the placental barrier while others are unable to do so.

Summary.

1. The agglutinin content of 355 samples of cord blood has been determined.

2. When the mother is of group O, the α' and β agglutinins appear more frequently in the cord serum than they do when the maternal group is A or B.

3. The complete agglutinogen-agglutinin A B O blood group pattern is found in only 38·8% of cord blood samples, the agglutinins being absent from or incompletely developed in the remainder.

4. Agglutinins incompatible with the child's red cells were found in four samples of cord blood. These were possibly immune antibodies produced in the maternal serum by group-specific substances from a secretor fœtus, which by virtue of relatively small molecular size were able to traverse the placenta.

5. An agglutinin which was incompatible with the mother's red cells was found in one sample of cord blood.

6. Analysis of the figures obtained suggests that the evidence favours maternal origin of agglutinins in the

cases in which they could, in relation to the blood groups of mother and child, be of either maternal or infantile origin.

Acknowledgements.

We wish to thank Dr. J. J. Graydon for the statistical analysis of the figures presented in this paper, the resident medical officers and nursing staff of the Queen Victoria Hospital for their wholehearted cooperation, and Professor F. M. Burnet for his helpful criticism.

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Reviews.

LIFE ASSURANCE EXAMINATIONS.

A BOOK on the conduct of life assurance examinations by E. M. Brockbank is based on the experience of the author, who has acted as local medical referee for over sixty companies.[1] He has examined fifteen thousand persons from adolescents to septuagenarians, and sets out his methods of examination as well as the principles which should be applied in assessing risks. He devotes nearly half of the book to "Impaired Lives" which includes those proposers whose prospects of longevity are not so great as those of the average "good risk", because of some defect in the family or personal history, or in the health or habits of the proponent at the time of examination. It is important for the medical referee to know whether the policy is for a whole life or for a limited number of years, because it is quite likely that a person with a slight defect can be accepted for a limited number of years when the risk would be too great for the natural expectation of life. This applies especially to proponents over fifty years of age. Special ages are dealt with and different factors needing special attention from the examining medical officer in the under twenty group as well as in the over sixty group are clearly set out. Whereas nephritis in any form precludes assurance, albuminuria is given a special section and the change of policy of insurance companies in the last twenty years is explained in the light of scientific facts which go to show that albuminuria even of a marked degree may have no bearing on the longevity of the proposer. With the frequency of surgical interference nowadays it is always wise to obtain the opinion of the surgeon in cases in which an organ has been removed before the risk is assessed. Under the section "Impaired Lives" the author covers a wide field, and pays attention to each physiological system as well as to certain pathological conditions, foremost among which are tuberculosis and malignant disease. Venereal disease also receives a chapter to itself, as does the important subject of assessing the risks of women proposers. Nowadays the latter form a great proportion of applicants and in the author's series reach 5%. In the opening chapters the author deals briefly and clearly with types of policy and definitions. All through the book the methods of approach and lines of reasoning are set out without padding or circumlocution, and in less than two hundred pages the author has given a great deal of useful information briefly and concisely expressed. This book should be at hand for every medical officer who undertakes the examination of a proponent for life assurance and the assessment of the risk.

[1] "The Conduct of Life Assurance Examinations", by E. M. Brockbank, M.B.E., M.D. (Victoria), F.R.C.P.; The General Practice Series; Second Edition; 1947. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 176. Price: 12s. 6d.

STAMMERING.

"STAMMERING: ITS NATURE, CAUSES AND TREATMENT", by Kate Emil-Behnke, is a somewhat naïve little production, the purpose of which appears to be to convince the stammerer that he can be cured by the adoption of the exercises and methods of breathing prescribed by the author.[1] These follow orthodox lines, and chiefly seek to achieve diminution of the tensions that exist in the muscles of the larynx, soft palate, tongue and lips and which thus lead to incoordination of respiration and articulation.

The chapter dealing with these exercises is the only one likely to interest the medical reader. The remaining chapters deal with the nature and causes of stammering, the "nervous factor", and general treatment. The author wisely points out the dangers of bullying, teasing or mocking the stammering child, an approach so often adopted by unwise parents. As the book is obviously intended for the lay reader, we should be ultracritical to detail the minor errors in the description of the physiology of respiration and speech production.

The author makes many *ex cathedra* statements which are not supported by any evidence. She claims that stammering is due to a shock to the sympathetic nervous system in childhood which deranges normal breathing.

Although there is a chapter devoted to the "nervous factor", the treatment of the emotional aspect of the disorder is wisely regarded as beyond the province of the book. Insufficient emphasis is laid upon the necessity for such treatment, however, the author merely stating that reeducation of the muscles should be "in conjunction with the treatment of the nervous factors".

For the lay reader, and especially for the stammerer, this book should prove interesting and helpful. It could be read to advantage also by the medical student.

[1] "Stammering: Its Nature, Causes and Treatment", by Kate Emil-Behnke; 1947. London: Williams and Norgate. Melbourne: Wyatt and Watts Proprietary, Limited. 7¼" × 4¾", pp. 100, with illustrations. Price: 6s.

SURGICAL PRACTICE.

IT is a pleasure to look upon and to handle Volume I of "British Surgical Practice", edited by Sir E. Rock Carling and J. Paterson Ross, with its beautiful binding and art paper.[2] The illustrations are well chosen and clear. The photography of clinical conditions and pathological specimens has been brought to a high degree of perfection.

[2] "British Surgical Practice", under the General Editorship of Sir Ernest Rock Carling, F.R.C.S., F.R.C.P., and J. Paterson Ross, M.S., F.R.C.S.; Volume I; 1947. London and Australia: Butterworth and Company (Publishers), Limited. 9¾" × 6¾", pp. 568, with many illustrations. Price: £3 17s. 6d.

The separate sections, written as they are by men particularly interested in the subject concerned, reach a high standard of excellence. However, there must be some lag between the writing of these articles and the launching of the completed volume, and this perhaps explains some omissions. Three of the contributors died before the appearance of the book.

Morley, in the first section, "Abdominal Emergencies", advises abandonment of the traditional double enema in suspected intestinal obstruction, a long overdue reform. He stresses that the viscera are insensitive, but Kellgren in the following section, "Abdominal Pain", states that the viscera vary greatly in their sensitivity. Kellgren gives an account of pains produced by the injection of hypertonic saline solution into interspinous ligaments and, somehow or other, derives therefrom conclusions about visceral pain.

Devine writes a good section on the "Abdominal Wall", but it is hard to understand how the medial fibres of the split rectus muscle can prevent the cutting out of sutures during a difficult closure of the posterior layer of rectus sheath. It is also hard to understand why lateral displacements of the right rectus ("medial" displacement is printed in error) should prevent a superadded incision to the left.

It would be of interest to know whether C. H. Bell really believes that a threatened abortion could be saved by vitamin therapy at such a late stage as he describes.

The chapters on achlorhydria and appetite, amyloidosis, abscess and adiposity might well have been omitted.

Acidosis is defined as a condition in which blood and tissue fluids "are more acid than they are in their normal condition", and "the conclusive proof of uncompensated acidosis is a fall in the hydrogen ion content of the blood". These are just slips. The factors influencing the choice between normal and fifth-normal saline solution are not discussed. Nor are the symptoms of water intoxication. Nor is sodium chloride mentioned as a factor in œdema. Rectal infusion is rated as "not advisable". But there are some occasions when this route may be very useful for the supply of water.

We should imagine that few would agree with Crymble when he writes in the section on "Adhesions" that "attacks of pain in the right iliac fossa following removal of the appendix are probably due to adhesions". Surely anxiety neurosis is the common cause.

Excellent chapters are given on "After-Care". Dr. Howland writes of physiotherapy above the "mobilised" region, where "immobilised" was intended. His omission of swimming from his lists of remedial games is probably a reflection upon the Toronto climate.

The section on post-operative care by a senior surgeon shows a sturdy conservatism. "Bronchial embolus" and atelectasis, water-salt balance, and vein ligation and anticoagulants for pulmonary embolism are not mentioned. Heparin is mentioned under thrombosis. It is suggested that further experience may show it to be a potent weapon, but no directions are given for its administration and "Dicumarol" is not mentioned. The traditional British surgical ritual of purgation before and after operation is adhered to, although its role in causing painful colicky spasms is admitted.

Under "Anæsthesia", controlled respiration is mentioned, but not discussed. Curare is not mentioned.

Under "Appendicitis", non-filling of the appendix after a barium meal is given as a pathological sign, in spite of the fact that many normal appendices fail to fill. The paramount importance of the pain shift from the centre to the right side in the differentiation of appendicitis from pneumonia, pyelo-ureteric colic *et cetera* is not recognized. Two types of "ileus" are described: (*a*) general peritonitis and (*b*) intestinal obstruction caused by localized peritonitis. In other words, the author realizes, possibly subconsciously, that there is no such thing as ileus, apart from peritonitis and obstruction. But he has not brought himself to the point of saying so. He shows how writers of textbooks retain dead wood out of respect for tradition. The same may be said of his rules for drainage. "When in doubt, drain" should be replaced by "Drainage of a dried field is damaging". Under complications (fistula, hernia *et cetera*) the ætiological relationship between the complications and the drainage tube is not mentioned. The opening of a pelvic abscess *per rectum* is not well described. A rectal speculum is not a good instrument for this purpose. The method described by Kirschner is better.

The section by Perkins on "Amputations" is easily the best that we have read on the subject, and it has an admirable sequel by Langdale Kelham on the management of "Artificial Limbs". So many of the other sections are excellent that space does not permit their special mention.

Under "Autonomic System" Paterson Ross states that "many patients have been relieved of intermittent claudication by sympathectomy", while Telford states that sympathectomy "will not cure claudication". A minor slip is Telford's statement that "the costo-cervical artery may be divided and ligatured in continuity". Nevertheless, these sections are excellent, and not the least important part is the well-reasoned warning given by Ross against the prevalent belief that sympathectomies are necessarily better because they are bigger.

Surgeons may well be proud to possess this beautiful and useful volume.

HYGIENE FOR NURSES.

THE ninth edition of the well-known book "Elementary Hygiene for Nurses" by H. C. R. Darling has recently been issued[1], and, as stated in the preface, "the whole work has been thoroughly revised and brought up to date". Throughout the book the author does not forget that the readers will be mostly trainee nurses, and so he does not burden their studies with any subject matter that is beyond their scope, but he writes clearly and dogmatically upon all the phases of the subject. Some chapters have been written for the book by writers who are acknowledged specialists on these topics, but most of the work is typical of the painstaking methods of the author. The chapters on food and on the dietetic treatment of diabetes are excellent, and so too are those on sanitation, but in such a book there is no need to particularize, and it is sufficient to say that this book should prove of value not only to trained nurses but also to tutor sisters and doctors whose duty it is to lecture and teach the subject of hygiene. Clear type printing and many fine diagrams and photographs adequately garnish the book and make it readily readable.

RHEUMATISM.

KENNETH STONE'S book on diseases of the joints and rheumatism is a very welcome addition to the literature on these subjects.[2] Stone is an honorary physician to the British Red Cross Society's clinic for rheumatism in Peto Place, London, and the fact that he can produce a book of this calibre is an argument in favour of a clinic for rheumatism. It is easy enough to say that there is nothing new in the treatment of acute and chronic rheumatism, but there is no doubt that a clinic of this kind collects human material, and at least the knowledge of the individual physicians attending the clinic should be enlarged. Stone states that this book is intended for students who regret the omission from the medical curriculum of teaching in the rheumatic diseases. This is an easy criticism to make, but what Stone may forget, or may not know, is that there is not time in two and a half years of the medical course to teach all students all about every department of medical knowledge from psychiatry to orthopædics. However, this book contains a useful summary of knowledge connected with joint diseases and rheumatism. There are chapters on the classification of joint diseases, on the anatomy and physiology of joints, on how to examine joints, and on all the different kinds of painful and diseased joints the author can think of. Rheumatoid arthritis and gout are fully dealt with, and the spine, the shoulders, the elbow, wrist and hand and the corresponding joints of the lower trunk are all discussed at length. Vagotonic rheumatism and fibrositis and pain in the back receive adequate attention. Treatment is considered under the headings of rest, exercise, drugs, electrotherapy and other headings in connexion with each type of rheumatism described.

This book is an excellent summary of up-to-date knowledge on rheumatism of the less acute kind; it does not deal with rheumatic fever.

[1] "Elementary Hygiene for Nurses: A Handbook for Nurses and Others", by H. C. Rutherford Darling, M.D., M.S. (London), F.R.C.S. (England), F.R.F.P.S. (Glasgow); Eighth Edition; 1944. London: J. and A Churchill, Limited. 7″ × 5″, pp. 299, with illustrations.

[2] "Diseases of the Joints and Rheumatism", by Kenneth Stone, D.M. (Oxford), M.R.C.P.; 1947. London: William Heinemann (Medical Books), Limited. 9¼″ × 6″, pp. 372, with many illustrations, some of them coloured. Price: 30s.

The Medical Journal of Australia

SATURDAY, MAY 29, 1948.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE PLACEMENT OF DISABLED WORKERS.

No reader of this journal can complain that he has not been kept informed of the different aspects of the problem associated with the rehabilitation of workers who have been disabled by war-time injury, by industrial accident and by illness. This is one of the subjects of social medicine that must never be neglected, firstly because the hazards to which men and women are exposed are continually changing or being added to, and also because industry left to itself is liable to lose sight of its complete responsibility to the individual and through him to the whole of society. For the advantage of those whose medical duties are specially connected with rehabilitation of the disabled it may be useful to set out some of our references to this subject. In the issue of June 19, 1943, rehabilitation was discussed from the point of view of military personnel and special reference was made to medical officers of the three branches of the armed forces. On October 16, 1943, a discussion was devoted to a "Report of the Inter-Departmental Committee on the Rehabilitation and Resettlement of Disabled Persons" issued in London in 1943. On August 18, 1945, attention was drawn to a study by officers of the United States Civil Service Commission on the efficiency of "impaired workers" in government industry. On July 13, 1946, the document discussed was a report of the Medical Advisory Committee of Scotland on rehabilitation. Our last reference to the subject was on January 11, 1947, when the report of the Standing Committee on the Rehabilitation and Resettlement of Disabled Persons, issued by the Ministry of Labour and National Services, was considered.

Among the bodies whose interest in this subject has never flagged is the International Labour Organization. It has discussed the subject at International Labour Conferences and has published articles and reports in its *International Labour Review*, which emanates from Geneva. It will be remembered that the International Labour Organization, which originated in Geneva, the home of the League of Nations, is an association of nations, financed by governments and democratically controlled by representatives of governments, of managements and of labour organizations. Its object is to promote social justice in all countries of the world and it functions through its secretariat, the International Labour Office. The organization since 1946 has entered into relationship with the United Nations as a specialized agency. The *International Labour Review* for November-December, 1947, contains an article on the placement of disabled workers to which attention should be drawn. Before this is discussed it may be useful to recall the purpose of a rehabilitation programme which is to ensure that persons born with an infirmity, and persons who have sustained an injury or contracted a disease which will result in permanent physical impairment, shall be made as fit as it is possible to make them and placed in gainful employment. The disabled person after treatment should be so placed that he will be able to compete on equal terms with his fellow man who is not disabled. This quite obviously will not be possible unless some consideration is paid to the mental outlook of the disabled person. In other words, rehabilitation must be concerned with the whole man and not merely with his stiff or deformed limbs, his crippled muscles, his defective eyesight or whatever the defect may be. With these general ideas in mind, we may turn to the article that has been mentioned.

We read that the aim of placement is "the employment of the disabled person in a job which he has the ability to do as efficiently as his able-bodied fellows without being a hazard to himself or jeopardizing the safety of the others, and without the possibility of aggravating his own disability". The process of placement should take into account the wide range in degree or extent of both the disabilities and the physical abilities and adaptability of individuals, and should vary in accordance with those differences. It should be unnecessary to state that in all dealings with individuals stress should always be laid on what a man can accomplish and that what he is unable to do should not be emphasized—the subject knows his own failings only too well and to dwell on them is quite likely to result in their aggravation and to cause mental distress. We are told in this article (which is unsigned) that every disabled person has special needs and special abilities and that it is obviously impossible to apply rigid, equal and mechanical rules. However, during the past few years some special methods and standards have been developed, and in several countries separate placement services have been organized on behalf of the handicapped as an integral part of the regular employment service machinery. In these services well-trained and specialized staff are employed. It is stated that the placement of the disabled calls for two types of adjustment. The first is a mental adjustment of the worker to his future job; the second is a correlation between the physical demands of the job and the physical condition of the candidate. Most of the special placement schemes or job analyses now used are apparently based on the second of the two types. But "both requirements must be fulfilled if failures are to be avoided, and it is hard to say which is the more important". This is the chief theme of the article and the point to which we would direct attention. Reference is made to systems that are in use in Canada, in the United States, in Great Britain, in France and in the Netherlands, but we need not concern ourselves with these. It will be more profitable to consider the "typical" system of psychological

testing that is described. It is divided into three main parts. The first test is designed to reveal the subject's range of interests and his work preferences—he is asked to indicate a list of occupations including those which he likes or dislikes. The choices may be used for the purpose of drawing conclusions about the individual's ability. The second test gives evidence of the subject's own self-evaluation; it shows the self-confidence, self-esteem and decisiveness which he possesses—he states whether he believes that he would be "good" or "poor" at managing specific situations. The third test, a *questionnaire*, consists of "a neuro-psychiatric and psychosomatic inventory of symptoms which are evidence of maladjustment". Certain crucial questions are known as "stop questions", because any subject who shows hesitation in answering them "must be stopped for more intensive appraisal". It is stated that the results of psychiatric interviews and procedures can be used in the obtaining of proper placement. This can easily be understood, but at the same time the question may be asked why this kind of investigation has to be dignified by being called "psychiatric interviews and procedures". This makes it all sound rather formidable and exclusive. Much more simply expressed is the need for "analysis of the work history of each case, in order to find out particulars of education, vocational training and previous jobs, if any". However, it is probably useless to quibble at words. Because a man has followed a certain occupation this does not mean that he should always stick to it. With growth in years and experience, such as that of the serviceman, horizons are widened and interests become more varied. Difficulty arises with disablement so that these new horizons and interests cannot always be followed or satisfied and it is then that the studied plan of mental adjustment may be adopted. To try to use this apart from full knowledge of the physical state of the subject is clearly absurd. One consideration remains—while we may conclude that it will generally be possible to find a place in industry for disabled persons, the attitude of industry to these persons must be friendly. By industry is meant industrial organizations or government departments on the one hand and individual able-bodied fellow workers on the other. It has happened that subjects, after undergoing long periods of vocational training and attaining mechanical efficiency, have been refused admission to the work for which they have been fitting themselves. Again, men are not always kind to those who appear to be handicapped, though they may really be quite fit. Plans of rehabilitation in industry will not really be effective unless everyone concerned wants to make them effective.

Current Comment.

STREPTOMYCIN SENSITIVITY IN NURSING PERSONNEL.

STREPTOMYCIN SENSITIVITY developed by those undergoing treatment with this antibiotic is not uncommon, but S. M. Rauchwerger, F. A. Erskine and W. L. Nalls have recently reported[1] development of sensitivity by six nurses engaged in handling streptomycin and have drawn attention to a small number of cases recorded elsewhere. The sensitivity in all six cases reported was characterized by an initial erythema followed by a most distressing pruritus and a papulo-vesicular eruption. Two patients had actual denuding of the skin over the terminal phalanges of the thumbs and index fingers of both hands—areas in contact with the drug during the handling of syringes and needles. Five patients had œdema of the eyelids with periorbital pruritus and photophobia, apparently due to local contamination of the area by the hands. The six nurses had been in intermittent contact with the administration of streptomycin for periods varying from at least five to fifteen months before manifestation of sensitivity, but it is pointed out that the actual sensitivity state may be produced in the absence of manifestations, leaving the possibility of a later outbreak of dermatitis and, more serious, of reactions that may prohibit the use of streptomycin in the face of urgent therapeutic indications. Rauchwerger and his colleagues were able to demonstrate the development of this sensitivity in appreciable numbers by means of intradermal tests carried out on a large group of nurses, the intention being to institute desensitization measures on those who reacted. They urge the desirability of intradermal testing in all institutions where streptomycin is used at all extensively, and also the use of prophylactic measures, such as the wearing of rubber gloves, with hand washing before and after the handling of streptomycin and of the syringes and needles used in its administration. They also consider it wise to allow to cool before opening sterilizers in which have been sterilized needles and syringes used with streptomycin, in case of "inadvertent nebulization". This hazard of streptomycin sensitivity applies not only to nurses, but also to pharmacists, laboratory technicians and others and may well involve other antibiotics. It presents a problem not yet fully solved and warrants careful consideration.

[1] *The Journal of the American Medical Association*, February 28, 1948.

AEROSPORIN: A NEW ANTIBIOTIC.

THE latest entrant to the field of antibiotics, aerosporin, is the product of *Bacillus aerosporus*, an aerobic spore-bearing bacillus of wide distribution whose natural habitat appears to be the soil. Although it is too early to draw any decided conclusions, two preliminary reports which have appeared[1] are encouraging. In the first of these, George Brownlee and S. R. M. Bushby, who with G. C. Ainsworth described the isolation of the antibiotic last year, have presented the results of in-vitro and in-vivo experiments with it in the laboratory, and in the second P. N. Swift has reported its use in the treatment of ten children suffering from pertussis. Aerosporin is selectively active against Gram-negative bacilli, so that its chemotherapeutic properties were mostly compared with those of streptomycin, but it is more discriminating in its activity than streptomycin. Its effect appears to be limited to a few Gram-negative pathogens, against which it is from ten to many hundred times more active than is streptomycin. There is no evidence that it has any action against the tubercle bacillus. Particular attention was paid in these investigations to infections in which established chemotherapy has so far been largely unsuccessful, especially pertussis and enteric fever, and against these aerosporin appears likely to be effective; other infections of which the causal organisms are highly aerosporin-sensitive are those due to the colon bacillus, paratyphoid fever, pasteurella and salmonella infections, bacillary dysentery, and, in the veterinary field, *Hæmophilus bronchisepticus* infections of dogs and calf-scour of calves. Infections with *Vibrio choleræ*, *Brucella abortus* and *Pseudomonas pyocyanea* may also be responsive. The indications are that it is likely to be at least as effective as streptomycin in *Hæmophilus influenzæ* meningitis of man.

Aerosporin is bactericidal *in vitro*, its action being influenced by the amount of serum in the broth and by the number of organisms present, so that successful

[1] *The Lancet*, January 24, 1948.

therapy may depend on the maintenance of continuous adequate serum levels. It is not absorbed from the gut, from which it eliminates sensitive organisms. Given parenterally it disappears from the blood stream quickly; hence four-hourly maintenance doses are necessary. In the normal animal it is not detectable in the urine, bile or cerebro-spinal fluid in a biologically active form—an obvious disadvantage. In a comparison with streptomycin two important considerations arise, toxicity and the development of resistance by organisms. Aerosporin is more acutely toxic than streptomycin in corresponding dosage, but the blood aerosporin level corresponding to the minimal lethal intravenous dose is at least 75 times greater than the average therapeutic blood level, so that there is ample margin for safety. Aerosporin is non-hæmolytic and has about the same toxicity to leucocytes as penicillin. The development of resistance by organisms tested was brought about only with a good deal of difficulty—a pleasing contrast with streptomycin. Difficulty was experienced from the presence of contaminating toxic substances, one an antidiuretic principle, the other adversely affecting the renal tubules; the first, however, has not been found to affect man, and purification methods have eliminated the second.

In therapeutic experiments with animals it was found that aerosporin gave complete protection against *Hæmophilus pertussis* with elimination of the infection, whereas streptomycin did not give protection. Similar effects were demonstrated in experimental infections with *Salmonella typhi*, *Hæmophilus bronchisepticus* and *Bacterium coli*. The really important therapeutic investigation, however, starts with Swift's administration of aerosporin to ten children, aged one month to two and a half years, suffering from pertussis. There were early and late, mild and most severe manifestations of the disease, and two patients were moribund. In most cases a therapeutic effect was observed within the first forty-eight hours. Vomiting, apnœa, cyanosis and the frequency of paroxysms were first modified; whooping and cough were last to disappear. Patients in whom treatment was begun within a week of the onset of whooping enjoyed an uncomplicated recovery irrespective of the severity of the disease or the age of the patient. When treatment was not begun until a week after the onset of whooping the effects were less favourable; this is probably related to the question of secondary invasion of the lung. Two of the children died, but the evidence suggests that the pertussis infection itself had been controlled and did not contribute directly to their deaths. It is suggested that early pertussis infections, treated before secondary infection has taken place, may be expected to respond to aerosporin alone; whereas in later infections a combination of aerosporin and a sulphonamide may be more effective. However, as *The Lancet* comments, this work also shows the difficulty of evaluating any treatment for pertussis.

Aerosporin is not yet in commercial supply (the material for these investigations came from the Wellcome Foundation, Limited) and much remains to be determined with regard to dosage and possible side effects. These reports have been reviewed rather fully, however, because although conservatively expressed they offer real promise. If subsequent reports confirm this work, an important gap in the field of antibiotic therapy will have been filled.

THE SOLUBILITY THEORY OF SILICOSIS.

AN interesting summary of the history of silicosis has been made by E. J. King of the British Postgraduate Medical School as an introduction to a critical study[1] of the current solubility theory of the disease and its causation. He describes the recording about the middle of last century of the finding of hard gritty material in lungs of solicotic subjects and the development of the theory that the pathological changes were due to mechanical trauma inflicted on the pulmonary tissue by the minute sharp-edged particles of stone dust. This "mechanical" theory was challenged by Gardner's discovery in 1923 that the dust of silicon carbide, although comparable in hardness and sharpness, did not produce the fibrous reaction caused by stone dust; and it was abandoned when a series of workers up to 1933 produced various lesions such as acute inflammation and necrosis with amorphous silica, colloidal silicic acid and finely powdered quartz. This work, and the demonstration by Kettle in 1932 that the same quartz particles which readily produced silicosis in animals would no longer do so if they were first coated with a thin layer of iron oxide, led to the modern "chemical" theory that the fine particles of stone dust in the lungs are pathogenic, not because they produce a sort of microscopic trauma, but because something of a toxic nature dissolves from their surfaces. This toxic substance is thought to be silicic acid. It might be supposed then, as King points out, that the pathogenicity of any stone dust would bear a direct relation to the rate at which it will release silicic acid into solution, and, broadly speaking, this is usually so. There are, however, anomalies in the application of the "chemical" theory and King has investigated it in a series of careful experiments which he describes in detail. Many discrepancies between silica solubility and pathogenicity were noted. Quartz dust was found to be more soluble the smaller its particle size, a progressive diminution in solubility taking place with fractions of all sizes when they were subjected to repeated treatment with buffered Ringer solution. All kinds and fractions of quartz tested produced reticulin and collagenous fibrosis in the lungs of rats. The mixture of 1% of aluminium hydroxide with the quartz fractions lowered their solubility but did not prevent the formation of fibrosis. A powdered sandstone (70% quartz) had a very low silica solubility but a high pathogenicity. A powdered shale (8% quartz) had a low silica solubility, though higher than the sandstone, and produced little fibrosis. More silica and alumina were dissolved from the shale than from the sandstone in a long-term leaching experiment. An extremely fine amorphous silica ("20 angstrom silica") had a high degree of solubility, and was rapidly fatal to animals when injected in large amounts. In smaller amounts, its injection into the lungs produced no fibrosis. Olivine (a magnesium silicate) had a medium silica solubility in the fraction of the lowest particle size and low degrees of solubility in the fractions of large particle size. It produced only slight fibrosis in the lungs of rats, with no apparent difference of effect between fractions of different particle size.

These findings have been set out baldly to show the discrepancies existing between the solubility and pathogenicity of silica and, as King observes, it might well seem that the time has come when some other explanation must be sought of the fibrogenic action of silica than the release from it of a toxic substance. He points out, however, that the chemical theory has been the basis of some notable practical discoveries in the prevention of silicosis and is too useful to be discarded. The differences may be reconcilable, although in what way is not immediately apparent. King draws attention to the fallacy of confusing test tube findings with what takes place in living tissues. Perhaps siliceous dusts release soluble silica in a different way in cells from that which occurs in the test tube, and some dusts may liberate the silicic acid in a different state, which is more noxious than that from other dusts. Silicic acid absorbed from the intestine and excreted by the kidneys does no apparent harm; perhaps it is different from that released from the surface of a quartz particle engulfed in the body of a phagocyte. As a culminating "daring" hypothesis, King suggests that perhaps a "nascent silica" or something of the sort may be released and produce a particular effect on the cell protoplasm. As he points out, it is in order to let the imagination run freely in considering possible causes of pathogenesis, provided that all ideas are submitted rigidly to trial. By his apparently destructive criticism of the current theory, King has probably freed it from early encrustation, and it will be interesting to watch further developments.

[1] *Occupational Medicine*, July, 1947.

Abstracts from Medical Literature.

MEDICINE.

Innocent Heart Murmurs.

W. EVANS (*British Heart Journal*, January, 1947) points out that the mechanism by which innocent cardiac murmurs are produced is quite unknown, but it is nevertheless possible to recognize them on clinical grounds alone. Innocent heart murmurs are usually mid-systolic. It may not always be possible, especially in the presence of tachycardia, to establish by auscultation that a given murmur is in fact mid-systolic; but whenever a gap can be detected between the first heart sound and the murmur following it, it is proof of the innocence of the murmur. Mid-systolic innocent murmurs may be divided into four groups: those best heard when the patient is in the reclining posture (common, especially in young subjects, soft, disappearing on deep inspiration, appearing in the pulmonary area also when the patient assumes the reclining posture), those best heard when the patient is in the upright posture (fairly common, especially in young adults, soft, disappearing on deep inspiration), those best heard in the patient's left parasternal region (less common, loud but not harsh, occurring in older adults), and a "loud" variety (uncommon, diffuse, transmitted towards the axilla when the patient is in the upright and towards the base when in the reclining posture). Innocent heart murmurs may also occur fairly commonly in late systole.

Febrile Reactions to Pneumothorax Refills.

P. H. ANGLADE (*Revue de la tuberculose*, 1947) has treated patients who habitually exhibit a rise in temperature after pneumothorax refills by administering one milligramme of ergotamine tartrate the night before the refill. The febrile reaction did not occur after this treatment.

Spontaneous Hyperinsulinism.

WALTER CANE (*The American Journal of Digesive Diseases*, June, 1947) discusses spontaneous hyperinsulinism and presents a survey of 28 cases of this syndrome. The author submits the suggestion that the condition should be called Harris's syndrome in honour of Dr. Seale Harris, who first outlined its description in 1924. The overall case incidence of 1·9% was experienced in the author's series of cases. The cardinal symptoms of the syndrome are abdominal pain, weakness, cold sweating, tremor, dizziness and impaired consciousness. Glucose tolerance tests were performed in 20 of the 28 cases studied, the diagnosis in the remaining eight cases being made on the basis of a fasting blood sugar level below 65 milligrammes *per centum* with satisfactory response to treatment. It was found that the typical blood sugar fall occurred much earlier than is usually believed, the average deep fall occurring after three hours, and earlier falls being noted after two hours. Falls in blood sugar level below 20 milligrammes *per centum* were seen in patients on whom no subsequent surgery was indicated. None of the patients in this series were submitted to surgery. The extent and the speed of the fall in blood sugar level were important factors in the causation of symptoms. Abdominal pain occurred as a symptom in 89% of the cases, a fact which is thought to be due to the known effect of insulin on gastric and intestinal peristalsis, and on gastric secretion and acidity. Attention is drawn to the relative frequency of this syndrome in patients presenting "ulcer histories" and in whom no diagnosis of ulcer can be made. No causative relationship between hyperinsulinism and peptic ulcer was found.

Treatment of Diabetes Mellitus.

GEORGE E. ANDERSON (*The American Journal of Digestive Diseases*, May, 1947) presents a creed for the treatment of *diabetes mellitus*. He states that while hyperglycæmia *per se* has never been proved directly to cause a single one of the more grave complications of the disease, the degree of glycæmia is the only real yardstick to measure the adequacy of control of the disease. It is in a similar category to the blood urea content in the uræmic state, but urea is harmless, whereas hyperglycæmia is not. Persistent hyperglycæmia is destructive of the "end-organ" in carbohydrate metabolism, and non-recognition of this principle entails surrender to the very pathological condition which it is intended to correct. The physician may wishfully, if misgivingly, regard as satisfactory the nutritional state and sense of well-being of his patient; but he has sacrificed every vestige of potential improvement in the patient's own intrinsic insulin mechanism. Also, he has often converted his patient into an irreversible diabetic with heavy and increasing insulin needs.

Hypertension and Unilateral Kidney Disease.

J. J. LANGLEY AND ROBERT PLATT (*The Quarterly Journal of Medicine*, July, 1947) review the indications for nephrectomy in hypertension. It is stated that an excretion pyelogram should be made for all patients with hypertension under the age of forty-five years, and in all cases if there is a history of cystitis, frequency, dysuria, hæmaturia or renal pain. This should be followed by retrograde pyelography when necessary. This investigation should also be carried out on all patients who give no familial history of hypertension, since there is at least a three to one chance that in such cases it is secondary to renal disease, which may be unilateral. If evidence of unilateral renal disease is found, there will be surgical indications for nephrectomy in a third of these cases, and in the remainder a non-functioning kidney on one side with good function on the other is the most promising indication for surgical success. In all cases the total renal function should be within normal limits. Age by itself is not a contraindication to operation, nor is the fact that hypertension has existed for many years. However, younger people are associated with a better surgical prognosis. Papilloedema is not a contraindication to operation. However, a family history of hypertension in a middle-aged or elderly subject contraindicates nephrectomy, unless the evidence of unilateral renal disease is abundantly clear. A history of pregnancy toxæmia has the same significance. The operation of thoracolumbar sympathectomy for hypertension affords an opportunity of inspecting the kidneys, and if unilateral renal disease is a possibility, the operation should be performed first on the side of the suspected kidney. A nephrectomy can then be carried out if the indications appear favourable, for example, atrophic renal changes, renal deformity, perirenal fibrosis, or abnormal renal arteries. In most cases of failure the operation of nephrectomy has been carried out when the evidence of renal disease has not been convincingly unilateral.

The Irritable Colon.

PAUL B. VAN DYKE (*The American Journal of Digestive Diseases*, June, 1947) discusses the recognition and treatment of the irritable colon. The basis of this disease entity is some unusual nervous disturbance to which is added some local irritation. The proctoscopic appearance is described by the author, which he regards as characteristic. Treatment is directed toward the neurogenic factor and toward the colon. The author's patients are admitted to hospital, put on a meat-free diet, and after four days a specimen of stool is examined for the presence of occult blood, ova and parasites. The patients are then given an ulcer type of diet consisting of bland food of *purée* consistency. Proctoscopic examination is carried out and a rectal smear examined. This is followed by a gastric analysis and a radiological examination of the whole alimentary tract. When the diagnosis of irritable colon is established, "Trilactic" is prescribed, the bland diet is continued, and sedatives are added to the *régime*. If thought advisable a psychiatric opinion is sought. Treatment of the neurogenic factor seeks to reestablish the normal nerve balance to the colon, and treatment to the colon aims to restore the normally low pH of the colon.

BAL in the Treatment of Agranulocytosis.

HOWARD L. HOLLEY (*Annals of Internal Medicine*, August, 1947) discusses the use of BAL in the treatment of agranulocytosis following intensive arsenotherapy for syphilis, and reports the results of this treatment in twelve cases. There were no deaths in the series, and the results clearly reflect the therapeutic action of BAL, and indicate the necessity for both prompt and adequate treatment in order to decrease the mortality in this complication occurring in arsenical therapy. The author suggests that BAL may decrease the time that is required for the bone marrow to return to its normal function, through its selective affinity for the suppressing agent. BAL was given in doses of 1·5 millilitres of 10% solution every six hours for forty-eight hours; then 2·0 millilitres daily for six doses. The drug is dispensed in a stable solution, peanut oil being used with benzyl benzoate as a solvent, and is administered parenterally. To three patients of this series multiple transfusions also were administered. In all cases the following adjuvant treatment was given: 5% dextrose solution given intravenously

to maintain the water balance; sodium perborate mouth washes to decrease secondary infection; and vitamin therapy. Also, in some cases, "Pentnucleotide" and liver extract were given. The author states that BAL is not an innocuous drug, but, if the dosage level of under three milligrammes per kilogram is adhered to, toxic reactions rarely occur. The toxic signs and symptoms of the drug are nausea, vomiting, headache, generalized aches and pains, burning sensations in the mouth, nose and eyes, sweating, restlessness, pain in the limbs, joints and trunk muscles.

"Trophic" Ulcers of the Hands after Myocardial Infarction.

E. SHAPIRO, M. L. LIPKIS AND JULIUS KAHN (*The American Journal of the Medical Sciences*, September, 1947) state that "trophic" ulceration of the hands is a rare and apparently hitherto unreported complication of myocardial infarction, caused, they believe, by impulses from the infarcted heart mediated antidromically through the sensory nerves. They suggest that antidromic impulses may be the mechanism of production of the other maladies of the shoulder and hand which follow myocardial infarction.

Intrathoracic Cysts.

E. W. DAVIS (*The Journal of the American Medical Association*, September 27, 1947) discusses intrathoracic gastric cysts. Congenital intrathoracic mediastinal cysts may be classed as dermoid, teratomatous, pericardial, simple, lymphangiomatous, bronchial and alimentary. The last named may arise from œsophagus, stomach or intestinal derivations. Only 25 alimentary cysts have been recorded. The author describes such a condition in a girl, aged fourteen years, whose main symptom was pain in the left side of the chest. There was slight dyspnœa on exertion, but no other signs. X-ray examination revealed a large, dense shadow of smooth outline filling the left side of the chest in its upper part. The usual clinical signs of tumour were present, with compression of the trachea and blocking of the main bronchus of the right upper lobe. At operation the mass was thought to be solid until it burst, with discharge of 1000 millilitres of greenish black fluid. The cyst was removed. Pathological reports disclosed a cyst lined with gastric mucosa.

Bronchogenic Carcinoma.

WILLIAM A. HENKIN (*Annals of Internal Medicine*, August, 1947) presents a study of 36 cases of bronchogenic carcinoma. He states that the rising incidence of bronchogenic carcinoma, together with the possibility of surgical cure by pneumonectomy, demands the earliest possible diagnosis. This disease may soon rank first among malignant conditions causing death in the male, and its diagnosis must be considered in any individual past thirty years of age with symptoms or signs referable to the chest, with low back pain, with suspected cord or brain tumour, or with bizarrely located metastases. X-ray examination of the chest suggests the correct diagnosis relatively early in about 60% to 75% of cases, and routine early bronchoscopic examination in every suspicious clinical syndrome is vital to early diagnosis and increases the percentage of lesions proved by biopsy. Fever, leucocytosis, anæmia and other evidence of serious pulmonary suppuration not infrequently characterize the clinical course of bronchogenic carcinoma. The average life expectancy at the present time is only eight months from the onset of symptoms to death. Demonstrable bone metastases can be found by X-ray examination in more than 50% of cases. Every patient without metastases or evidence of local invasion of vital structures should have the benefit of exploratory thoracotomy. X-ray therapy is merely palliative, but it should be given as early as possible after the determination of non-resectability.

Erythema Nodosum.

CUTTING B. FAVOUR AND MERRILL C. SOSMAN (*Archives of Internal Medicine*, October, 1947) discuss the ætiology of *erythema nodosum* and review the related literature. An analysis is presented of the clinical features of this disease in 155 patients, 61 of whom were under observation for one to twenty years. The age incidence was from three years to sixty-six years, the preponderance of occurrence was among women (86%), and the disease usually developed in the winter months. Associated respiratory infections accompanied 80% of the attacks, and β hæmolytic streptococci were grown in culture from throat material in one-half of the cases. Tuberculosis was an uncommon antecedent infection and rheumatic heart disease was a rare sequela. Migratory polyarthritis was a part of the clinical picture of *erythema nodosum* in 80% of adults and in a third of the children. Isolated attacks of pains in the joints preceded and followed the disease. In 10% of the patients a chronic form of the disease occurred lasting from months to years. Mild secondary anæmia, cervical adenopathy, and occasionally enlarged hilar or bronchial glands characterized the illness. *Erythema nodosum* is thought to be a hypersensitivity disease, and individual predisposition, a variety of infections and chemical agents, and local trauma contribute to its occurrence. It is not a form of rheumatic fever.

Blood Pressure Readings of 75,258 University Students.

RUTH E. BOYNTON AND RAMONA L. TODD (*Archives of Internal Medicine*, October, 1947) present the results of blood pressure readings on 75,258 persons, of whom 43,800 were men and 31,458 were women. Most of the subjects were under the age of twenty-six years and the data for the series were derived from the records of physical examinations made at the Students' Health Service at the University of Minnesota from 1930 to 1942. The blood pressure was recorded by the auscultatory method, with a mercury manometer, and the subject in the sitting position. The diastolic reading was taken at the point at which the tone changed. The mean systolic blood pressure for men of all ages was 122 millimetres of mercury, and for women 111 millimetres; the mean diastolic pressure for men was 74·5 millimetres of mercury, and for women 69·7 millimetres. The mean systolic pressure of women showed a tendency to rise with age, but this was not noted in the case of the men. In both men and women there was a tendency for the diastolic pressure to increase with age. A greater percentage of women than men had systolic pressures below 109 millimetres of mercury, but systolic pressures over 120 millimetres occurred more frequently in men except in the age group of forty-one years and over. Approximately two-thirds of the men had systolic pressures between 110 and 129 millimetres, while about two-thirds of the women had systolic pressures between 100 and 119 millimetres. The sex differences in diastolic pressures followed the same pattern as in the systolic pressures.

Incidence and Clinical Significance of Coronary Artery Disease in Diabetes Mellitus.

SAMUEL STEARNS, MONROE H. SCHLESINGER AND ABRAHAM RUDY (*Archives of Internal Medicine*, October, 1947) discuss the incidence and clinical significance of coronary artery disease in *diabetes mellitus*. A study of the hearts of fifty diabetic patients by means of post-mortem injection and dissection technique was made; and in approximately three-fourths of these functionally significant coronary artery disease was demonstrated. One-third of these patients had died of acute coronary artery heart disease and one-quarter had suffered with *angina pectoris*. Among diabetic women over the age of forty years the incidence of significant coronary arteriosclerosis, of *angina pectoris*, and of death due to coronary artery disease is as great as among diabetic men. This is in sharp distinction to the sex difference in these respects in the non-diabetic population. *Angina pectoris*, deaths due to acute coronary disease and congestive heart failure all are more common when hypertension is present in diabetic patients than when the blood pressure is normal. The severity of the coronary arteriosclerosis was found to be correlated with the duration, but not with the severity, of the diabetes. The authors are of opinion that early detection of the symptoms of coronary artery disease in diabetics is necessary if the sequelæ of coronary arteriosclerosis are to be postponed or avoided. Mild hyperglycæmia is preferable to a regimen which may result in insulin hypoglycæmia and the attendant risk of precipitating myocardial infarction.

Genetic Predisposition and Virus Pneumonia.

EDWARD H. KOTIN (*Annals of Internal Medicine*, November, 1947) reports the occurrence of attacks of severe virus pneumonia in close succession in six genetically related female members of a family. Corresponding male members escaped infection. The author states that the occurrence suggests that a genetic predisposition to the disease existed in the subjects affected.

Penicillin and Lung Abscess.

EUGENE ROSENTHAL (*The Lancet*, December 6, 1947) states that penicillin given intramuscularly may be expected to produce improvement but not final cure of lung abscess. The method of choice appears to be the intrabronchial administration of penicillin supplemented by frequent inhalations. The author reports that of eleven patients treated by this method ten recovered.

Public Health.

REPORT OF COMMITTEE OF INQUIRY INTO CLAIMS OF CANCER CURES BY MR. J. BRAUND.

THE following is the text of the report of a committee of inquiry appointed by the Honourable James McGirr, M.L.A., Premier of New South Wales, to investigate claims by Mr. J. Braund to have cured or to be able to cure cancer. The report was presented to the Parliament of New South Wales and was ordered to be printed on April 8, 1948.

REPORT OF COMMITTEE OF INQUIRY INTO MR. J. BRAUND'S CLAIMS TO CURE CANCER.

The claim that Mr. Braund possessed a cure for cancer was first published in an article in *The Sun* on 3rd December, 1947, in which Mr. Braund is alleged to have claimed that he had "treated 318 Australians for cancer, and cured all but one of them". It was also alleged that "his one stated desire is to be officially and thoroughly examined by the Government . . ."

Reference was made in this and subsequent articles to the approval and support of a cancer expert who remained anonymous.

The original article was based primarily on the case of Mr. Kilpatrick, a farmer of Cassilis. Inquiry by the Health Department indicated that three other patients, in addition to Mr. Kilpatrick, had been communicated with by telephone by the newspaper.

It is necessary to deal briefly with these patients' histories.

Mr. Kilpatrick suffered from a bowel condition for which he was examined by X ray. The radiologist mentioned the possibility of cancer of the bowel and his medical advisers gave this diagnosis very careful consideration and perhaps some credence. Mr. Kilpatrick is quite convinced that he was given only six weeks to live and at this stage underwent treatment by Mr. Braund, who, it is alleged, removed the cancer by an injection in his back.

Subsequent events showed that Mr. Kilpatrick's condition was due to ulcerative colitis which causes an intense spasm of the bowel and such spasm seen in the X-ray photograph was regarded as a blockage possibly caused by malignant disease. Mr. Kilpatrick is still suffering from ulcerative colitis which he ascribes to the after-effects of cancer and resolutely refuses to concede the possibility of an error in diagnosis. This contention, he maintains, is an evasion by the medical profession to explain the phenomenon of his survival.

The other three patients (A.G.D., rodent ulcer; F.W., osteoarthritis, diagnosed as cancer by Dr. Brose; S.E., diagnosed by his doctor as circulatory disturbance of left leg and foot, "no cancer seen") were never diagnosed as cancer by any medical practitioner, but are firmly convinced that they have been cured of the disease.

Press publicity was intensified and demands were made in the Press, in Parliament and elsewhere for an immediate inquiry into Mr. Braund's claims.

On 29th January, 1948, the Premier announced "that the Government had decided to set up a Committee of Inquiry to investigate Mr. Braund's claims to cure cancer. It is proposed that the Committee will consist of four members nominated by the medical profession, the Director-General of Public Health (Dr. E. S. Morris) and three lay observers. Mr. Braund will be afforded the opportunity of nominating a number of medical practitioners equal to those nominated by the medical profession. The Premier added that the Department of Public Health had been carrying out an investigation of cases nominated by Mr. Braund, but as completion of these investigations would take some time, it is considered that, in the interests of the public generally, it would be preferable to establish the Committee immediately, and he therefore proposed to ask the Committee to proceed with its investigations with the utmost urgency."

On 4th February, 1948, the Committee met at the invitation of the Premier in the Executive Council Chamber, Chief Secretary's Department, when Mr. P. H. Roper, Under Secretary, Premier's Department, addressed members on the importance of determining the validity or otherwise of the claims put forward by Mr. Braund.

Those present as members of the Committee were: Dr. E. S. Morris (Chairman), Director-General of Public Health; Dr. B. T. Edye, surgeon, Dr. A. J. Collins, physician, Dr. A. H. Tebbutt, pathologist, Dr. A. T. Nisbet, radiologist (nominated by the B.M.A., N.S.W. Branch); Dr. M. Barry, Dr. G. H. B. Deck, Dr. H. V. Gillies (general practitioners appointed by the Premier on behalf of Mr. Braund); H. L. Brose, Esq., D.Sc., Ph.D. (lay observer appointed by the Premier on behalf of Mr. Braund); V. H. Treatt, Esq., M.L.A., C. C. Lazzarini, Esq., M.L.A., E. J. Hallstrom, Esq. (lay observers).

The Committee at once entered upon its investigations.

Efforts to Examine Patients Alleged to have been Cured of Cancer by Mr. Braund.

Prior to the appointment of the Committee, the Chairman (Dr. E. Sydney Morris) in his official capacity of Director-General of Public Health, had collected information concerning certain patients and this information was submitted to the Committee.

In this way the names of sixteen patients who had been treated by Mr. Braund came under the notice of the Committee at the outset of its investigation.

Some were persons who had written to the Department alleging they had been cured of cancer; some had called voluntarily; and others were submitted to the Committee at its first meeting by individual members.

Subsequently Mr. Braund submitted a list containing the names of twenty-one patients, several of whom were included in the sixteen cases first mentioned. The remainder were scattered all over New South Wales, Victoria and South Australia, and, in consequence, not readily accessible.

The Committee, having considered the histories available of the sixteen cases, were of the opinion that ten had suffered from non-cancerous or doubtful conditions and that six were probably suffering from cancer. An effort was made to examine these six patients, but only one submitted to examination. Several intimated that Mr. Braund was not agreeable to their examination at that time.

The Committee at this stage appeared to have reached an impasse, since it was not possible to proceed owing to the reluctance of patients to be examined.

However, arrangements were made whereby Mr. Braund interviewed the Committee. The interview took place on 24th February, 1948. At the interview Mr. Braund agreed to advise the patients to submit themselves for examination and undertook to forward the names and addresses of six additional patients whom he had cured of cancer.

In due course Mr. Braund submitted the names of thirteen additional patients, one of whom (Mr. C.) resided in Victoria; one (Mr. B.) had only commenced treatment; two (Mrs. S. and Mrs. H.) were of doubtful diagnosis; one (Mr. C.) was a rodent ulcer; two (Mr. T. and Mr. W.) had not been contacted when investigation terminated; one (Mrs. S.) died from cancer before the Committee could see her; one (Mrs. L.) at first agreed to be examined, but finally refused; and only four (Mr. W., Mrs. E., Mrs. M. and Mr. W.) were actually examined.

Of the six patients selected by the Committee at the outset of the investigation, one (Mr. G.) resides in Victoria and intimated he was too ill to travel; three (Mrs. F., Mrs. J. and Mrs. M.) refused to be examined; and only two (F.P. and Mr. R.) were actually examined by the Committee.

Every facility was given to enable patients to attend for examination. Motor transport to and from the hospital was provided and in the case of the patient who resided in Victoria an offer of all rail, air or other fares plus a travelling allowance of £1 10s. per day was made.

The Committee has every reason to believe that most, if not all, of the patients who refused to come forward for examination did so on the advice of Mr. Braund.

Demonstration by Mr. Braund of His Treatment.

At his interview with the Committee on 24th February 1948, Mr. Braund agreed to the medical members of the Committee visiting his home on 1st March, 1948, in order that he might demonstrate his method of treatment, especially the technique of his injection. This arrangement was subsequently confirmed in a letter to Mr. Braund from the Chairman of the Committee.

The visit was made on the appointed day. Mr. Braund demonstrated his method of what he termed "restoring the circulation" by having the patient place his feet and legs in a cylindrical galvanized iron container which contained hot water to approximately just below the knees. After a few minutes the patient was placed on the bed and Mr. Braund proceeded to massage the toes by his special method.

He explained that his ideas differed from the orthodox view in that the orthodox conception was that blood should be pressed back towards the heart, whereas he believed in forcing the blood from the heart through the tissues. To effect this he rubbed and squeezed each side of the toes, particularly the great toe, emphasizing that the blood was being forced through the gland alleged to be at the side of

and near the tip of the great toe. It may be mentioned in parenthesis that no such gland exists. Later he used a small towel stretched across the front of the leg and pressed firmly downwards from the knee towards the ankle.

He claimed to be able to reduce the pulse rate within a few minutes by massaging the toes even if the patient were in an epileptic fit and also to reduce the blood pressure in some similar manner.

The impression made on the minds of the medical practitioners present was that Mr. Braund's conceptions of anatomy and physiology were entirely erroneous and contrary to proved and universally accepted scientific facts.

Mr. Braund did not demonstrate his injection technique, excusing himself by stating that his patients had become frightened and had departed. Members of the Committee were disappointed, but felt that this was only another instance of lack of frankness and cooperation on the part of Mr. Braund.

Mr. Braund showed two female patients who had recently been treated by him by his injection method.

One (Mrs. L.), suffering from inoperable cancer of the *cervix uteri*, had been under his treatment for some time and had been injected near the lumbo-sacral junction of the spine on 25th January, 1948. A slough, alleged to be the cancer, was removed on 15th February, 1948, and at the time when seen by members of the Committee the patient showed a purulent ulcer approximately two and a half inches in diameter and three-quarters of an inch deep at the site of injection.

The other patient (Mrs. A.G.) showed a large hard mass more or less infiltrating the whole left breast. Glands were palpable in the left axilla, and in the neck above the left clavicle. The condition was regarded as probably cancer. As the patient could only speak Italian it was not possible to obtain a full history. Mr. Braund had injected the breast some time previously, and in the presence of the Committee removed a piece of necrotic tissue (about two inches square and half an inch thick), allegedly the cancer, which, on request, he gave to the Chairman of the Committee.

Immediately on returning to the city action was taken by the Chairman to have the tissue examined microscopically. The report in connection therewith is dealt with below.

The Diagnosis of Cancer.

It has been already mentioned that the majority of patients whose names were submitted to the Committee as having been cured of cancer had not been diagnosed by a medical practitioner. In most instances the diagnosis had been made by the patient himself, by Mr. Braund, by Dr. Brose, who is not a medical practitioner, or other lay person, and, naturally, the patients thought they were readily cured of the disease from which they were not suffering.

Some patients suffering from rodent ulcer were definitely convinced that their condition was typical malignant cancer, and, in view of the importance of this misinterpretation, it is considered advisable to explain why such cases cannot be accepted as a test of any claim for cure of cancer. Rodent ulcer (basal cell carcinoma) is a very special variety of localized tumour. It commences as a papule or nodule in the skin, which if untreated gradually extends, and as it extends the central portion breaks down, forming an ulcer. The condition is usually painless, neighbouring lymphatic glands are not involved, no metastases (secondary deposits) occur in other parts of the body and the general health does not suffer except in the later stages perhaps after many years.

The condition is readily curable in its early stages, but if neglected over many years can cause extensive local damage.

The important point, however, is that rodent ulcer does not constitute the "cancer problem" as understood by the scientific world. The cancer problem signifies those forms of malignant disease which are liable to recur in spite of all known methods of treatment and to give rise, perhaps several years later, to metastases (secondary growths) in other parts of the body which usually bring about a fatal termination.

The diagnosis of cancer is by no means a simple matter, and in the case of internal organs may be extremely difficult.

The only certain test that a tumour is a cancer is proof by microscopic examination.

No matter what clinical evidence may justify the diagnosis of cancer, especially in the case of internal organs, the conclusive and final decision can only be made by microscopic pathological examination of the tumour.

It is for this reason that, in assessing any claim for the cure of cancer, it is essential to be quite certain that the patient has suffered or is still suffering from that disease.

Although one or more medical practitioners may feel justified in arriving at a diagnosis of cancer on clinical evidence, there is always the possibility that such diagnosis may be mistaken in the absence of proof by the microscope. Unfortunately, once the possibility of cancer is mentioned, the patient may become convinced that he is doomed, and, discarding medical treatment, seek out some alleged "cure".

If a mistake in diagnosis has been made, the patient is subsequently proud to tell the world that he has been cured of cancer by some vaunted panacea.

Towards the end of the investigation, Mr. Braund wrote to the Premier—and incidentally published at the same time the import of his letter in *The Sun* of 19th March, 1948—complaining that the Committee was unfairly selecting only serious and advanced cases for examination.

The Committee wishes emphatically to state that all the patients who were examined had been nominated by Mr. Braund or had been approved by him. The Committee would have readily examined a number of other patients but for the lack of cooperation by Mr. Braund, who, after agreeing to their examination, advised the patients not to attend.

The Committee made no distinction between patients on a basis of the seriousness of their condition or the type of cancer with which they were afflicted.

The only criterion was that the patient was suffering from some form of cancer and that that fact was established beyond all reasonable doubt.

On the other hand, Mr. Braund appears to claim for himself the right to diagnose cancer by special intuition without any ancillary assistance such as are provided by X rays and the microscope.

Judging by the histories of many patients, it seems that any lump or swelling on the skin surface, whether it be a mole, sebaceous cyst or simple fatty tumour, etc., is diagnosed as cancer by Mr. Braund.

Naturally such patients become in time living examples of the efficacy of Mr. Braund's alleged cure for cancer.

The publicity associated with Mr. Braund's claim has, undoubtedly, established in the public mind the idea that he possesses a specific cure for any type of malignant disease embraced by the term cancer.

In his interview with the Committee, however, Mr. Braund was much more guarded in regard to his claim as shown by the following verbatim notes:

DR. MORRIS: You sent me twenty-one names and some are scattered all over New South Wales, Victoria and South Australia. Then out of the whole lot that have come under my notice there were only sixteen, concerning whom I can get information from the doctors. Of these sixteen, five were definitely cancerous and ten were very doubtful, or not cancerous. We want to see those five or six cases which we are satisfied are cancerous, and which you say you have treated.

MR. TREATT: That seems reasonable.

MR, BRAUND: They are six of the worst cases. I would not like to convey that I would cure them. I would not like to express that to any of you unless I know. How could I?

DR. MORRIS: Three were on the cinema film and we presumed you knew they were there. I understood you said you had taken their cancer away. We will get the films shown for you if you like.

MR, BRAUND: Which are the names you mentioned?

DR. MORRIS: Mr. R., Mrs. J. and Mr. G. They all said they had been cured of cancer by you.

MR. BRAUND: Mrs. J. could not have said that.

DR. MORRIS: She said Mr. Braund had taken away the cancer without hæmorrhage, without an anæsthetic and without a knife. If that does not amount to saying you have cured her of cancer, I do not know what it is.

MR. BRAUND: Mrs. J., I took the cancer away. I thought you were going—you saw it.

DR. MORRIS: I want the Committee to see Mrs. J.

MR, BRAUND: Give me the five; I will note them off.

DR. MORRIS: Mrs. J., Mr. R., Mr. G., Mrs. F., Mrs. M.M.

And again:

MR. LAZZARINI: I have met Mr. Braund a few times, and I have become interested through ———, whom he was treating. I know when he was asked to submit these cases he said: "Yes, I will submit them, but they are putting a difficult job up to me. They are all cases the doctors treated by one method and they have been unsuccessful cases. They are putting very hard cases up to me, and that is the point." He said: "There are none of these cases who have come to me direct and I have started my treatment *de novo*. They are all cases the medical profession have had for treatment and there has been a failure."

DR. COLLINS: The point is, Mr. Braund does not exactly make the claim he can cure all types of cases.

MR. LAZZARINI: No, but he hopes to.

DR. COLLINS: That is something which I did not know before, and I am glad to hear it now.

The Committee was not concerned in its investigation with simple (non-cancerous) or, as they are termed, benign tumours. Such tumours may be present for many years and may have little or no effect on the patient's health. Their treatment offers no difficulty and they usually do not constitute any danger to life.

Such a tumour can be removed in various ways, including the injection around it of an escharotic agent, though the latter method would result in unnecessary disfigurement.

To contend that the removal of a simple, benign tumour by means of an escharotic constitutes a cure for any form of cancer is either a fallacy based on misconception or an intentionally misleading claim.

Prognosis of Cancer.

The dread with which such a serious disease as cancer is naturally regarded is liable to cause the patient to believe that his expectation of life is only a matter of a few weeks or months. This belief is sometimes based on the grave view taken by his medical adviser, though such view, like all human judgements, is not infallible.

Although the outcome may be inevitable, it is not possible to be certain in any case of the prognosis in point of time. The various types of cancer differ in regard to their degree of malignancy; but so many factors are involved that dogmatic estimates of the expectation of life are rarely justified. In some instances the patient may live for a comparatively short period; in other instances he may live for several years, but some are completely cured.

Dr. Ralston Paterson, a recognized world authority on cancer, on his visit to Australia, made the statement that 42 per cent. of all cancer patients admitted to the Institute at Manchester for surgery and radiotherapy lived over five years.

Mr. Braund now contends that where his treatment is not entirely curative, it will, nevertheless, prolong the patient's life.

The Committee has seen no evidence to support this contention, and, in the light of the facts abovementioned, considers that such contention is unjustified.

Does Surgical Operation or X Radiotherapy Interfere with Mr. Braund's Treatment?

Mr. Braund contends that his treatment is jeopardized, if not actually rendered abortive, when it is given subsequent to treatment by surgery or X radiotherapy. Such a statement may impress a lay person, and, no doubt, affords a ready explanation why an alleged specific cure fails in any given case.

The Committee wishes to state as emphatically as possible that there are no grounds whatever to support this false notion. If Mr. Braund's treatment were a specific cure for cancer, as alleged, its action would be facilitated or at least assisted by the removal of the main source of cancer cells in the primary tumour.

At least one patient (case history I) was treated by Mr. Braund from the outset. She was subjected neither to surgery nor X radiotherapy, and yet he failed even to relieve the condition.

Cases Treated that have No Positive Evidence of Cancer.

Several patients without cancer have been seen, and many more have been described to the Committee. One reported case was apparently that of a ganglion (a cyst in the tendon sheath) on the back of the wrist. This supposed cancer was injected by Mr. Braund, and claimed as a cure. In days gone by the method of cure was to hit the swelling sufficiently hard with the family Bible to cause bursting of the wall and a subsequent cure.

Cases that have been Diagnosed as Cancer (as far as is Possible without Microscopic Examination) and who with Ordinary, Modern Forms of Treatment would have a Reasonable Chance of Cure.

Two such cases were seen by the Committee, and in both cases it was considered that the longer they remained under this unusual treatment, the less chance did they have of living. Under such circumstances it appears that Mr. Braund was materially assisting in the death of these people.

Cases of Advanced Cancer in which Medical Science at Present Realizes that Cure is Impossible.

Here Mr. Braund's treatment is subjecting these people to unnecessary travelling, to destruction of healthy body tissue, the dressings and otherwise unnecessary nursing attention. The expectation of life is in no one case increased, and may even be diminished. In such cases a certain psychological euphoria is produced which, of course, can only last a certain number of weeks. The resultant mental disappointment, terrible as it must be, seems hardly to warrant the temporary disillusionment.

Assessment of Mr. Braund's Treatment.

As outlined above, Mr. Braund's treatment consists of massage and hydrotherapy with the view of "restoring the circulation"; and when the patient's condition is judged to be satisfactory a local injection of some substance is made into a selected portion of the patient's body.

Although the number of patients examined has not been large, it has included cases typical enough of cancer with varying manifestations.

We have seen sufficient of Mr. Braund's methods to visualize a complete picture of the nature of his treatment. His hydrotherapy and massage treatment cannot possibly have any beneficial effect upon the progress of cancer. His methods are based upon unsound physiological and grossly incorrect anatomical bases. They betray his complete lack of comprehension of the nature of cancer and of its effect upon the body. If any apparent benefit has resulted in certain patients it can be attributed, without any doubt, to its psychical effects which are seen in certain patients upon varying therapeutic procedures. Precisely similar benefit may follow an exploratory operation for incurable abdominal cancer, at which the surgeon finds he can do nothing and closes the abdomen without doing more than examining the unremovable cancer. Such temporary benefit of psychical origin never deceives the trained medical mind.

The essential treatment, which forms the basis of Mr. Braund's claim, is the injection. The Committee was not given an opportunity, as promised, to see Mr. Braund actually inject his secret cure into a patient, nor did he divulge to them what he injected. It is a secret remedy. The medical members of the Committee, however, have seen the results of such injections and can readily visualize the technique.

The injection treatment consists of injecting an escharotic fluid into the subcutaneous tissues. The ulcers resulting from this injection are shallow and indicate that the fluid has a localized necrosing action. All such escharotic fluids coagulate the albumin of the body tissues and fluids, occlude the blood vessels and are not absorbed into the general circulation. It must be noted here that Mr. Braund does not inject the fluid directly into the blood stream. There can be no question, therefore, of such fluid having any remote action on parts of the body other than the site of injection. The site of injection may be some considerable distance from the actual cancer. For example, the patient is injected over the sacrum (termed by Mr. Braund the "genital vertebra") or perhaps the lumbo-sacral junction for cancer of the womb or lower bowel, over the lower dorsal vertebræ for cancer of the stomach, or in the abdominal wall for cancer of the liver. Within two or three weeks from the time of injection a slough of necrotic tissue is removed from the site of injection, leaving a purulent ulcer varying in size up to two and a half inches in diameter and about half an inch to three-quarters of an inch in depth. Further injections may be made from time to time. The ulcer gradually heals during several weeks with the formation of scar tissue.

The slough removed from the breast of Mrs. A.G. on 1st March, 1948, was submitted for histological examination by Dr. E. L. Morgan, Principal Microbiologist, whose report states:

> Sections show necrotic tissue which is mainly fibrous tissue containing degenerated cells amongst which can be recognized pus cells and red blood cells.
>
> The tissue is too degenerated for recognition of further histological elements.

The removal of the slough is claimed to be, and is certainly regarded by the patient as, the removal of the cancer. That a cancer, say, of the womb, many times the size of the slough can be removed through several inches of bone, muscles, etc., is absurd. It is contended on behalf of Mr. Braund that the substance injected seeks out the cancer cells wherever they may be in the body and destroys them *in situ*. In other words, the formation and separation of the slough from the body indicates that the cancer has been destroyed. The falsity of this theory can be seen in the histories of patients reviewed in this report.

Further, this theory, though very plausible, is not supported by any scientific evidence whatever, and is opposed to all known facts of the pathology of cancer.

There are numerous substances which, if injected into the body, will produce necrosis with separation of a slough similar to that produced by Mr. Braund's technique.

Acids, alkalis, arsenic, caustic and astringent chemicals, basic metals such as zinc and lead and many other substances are well-known escharotics which have been used for this purpose. Mr. Braund's treatment is not unique, since he has had numerous precursors in his special field.

In 1932 the "Roberts Cancer Cure" was launched with great Press publicity in Queensland, and a public company was formed with considerable capital to exploit what was claimed to be an absolutely certain cure for cancer.

The essential part of the Roberts cure was an injection which produced necrosis in a similar manner to Mr. Braund's method, though, no doubt, the escharotic employed may have been different.

In 1933 an expert committee appointed by the Government of Queensland submitted a report on the "Roberts Cancer Cure" and a copy of the report is attached as Appendix 1.

About 1936 the "Koch-Baker Treatment" was introduced into Australia from U.S.A., where it had been denounced some few years previously.

In 1938 the "Koch-Baker Treatment" became the subject of inquiry by a committee appointed by the Government of New Zealand. A copy of the committee's report is attached as Appendix 2.

There is perhaps no need to say more than that there is a striking similarity between these treatments and that employed by Mr. Braund. The technique is similar, the resulting necrosis is similar, the theory on which they are based is similar, and, unfortunately, the outcome for the patient is likewise similar.

The Committee is definitely of the opinion that Mr. Braund's treatment is in no way a specific cure for cancer as claimed by him, nor can it retard the growth of cancer.

Case Histories.

NOTE.—All patients comprised in the following case histories, with the exception of Case 1, were nominated by Mr. Braund as patients who had been cured by him or were approved by him for investigation on the nomination of the Committee.

The following patients were examined by the medical members of the Committee:

CASE A.—Male; age about 30 years. Malignant mole. Had two moles in left temporal region excised in October, 1946. In August, 1947, a nodule appeared below left ear which was excised. Then two nodules appeared in pre-auricular glands and a radical operation was performed to remove the tumour in front of ear, together with portion of the parotid gland and the upper cervical lymph glands. On 8th December, 1947, there was evidence of recurrence in the upper deep cervical glands on left side.

Pathological report: "Sections show massive deposits of a malignant melanoma."

On 16th December, 1947, Mr. Braund injected the tumour and a fortnight later removed a slough. This received much Press publicity and was hailed as a complete removal of the cancer. The patient had three subsequent injections prior to examination by the Committee on 20th February, 1948, when there was a large mass in the upper cervical region, three inches in diameter, involving deeper structures. The mass was quite fixed and on its surface there was a sloughing area one inch in diameter, the site of the last injection. No enlarged glands detected elsewhere in neck. One small doubtful gland in left axilla. X ray normal.

Comment.—The patient is not cured. In the opinion of the medical members of the Committee he has not received any benefit from Mr. Braund's treatment.

CASE B.—Female; age 41 years. Radical amputation of right breast for cancer in 1944, followed by deep X-ray treatment. Recurrences occurred in wound in 1946 and 1947, which were controlled by deep X ray and radium. Patient commenced treatment by Mr. Braund on 9th January, 1948. Treatment consisted of twelve hot water and massage treatments followed by an injection into the left breast on 19th February, 1948. Nothing had been removed from the site of the injection up to 5th March, 1948, the date on which the patient was examined by the Committee.

Examination showed three or four small subcutaneous nodules between scar of operation on right breast and the sternum. The left breast is diffusely enlarged and firm, but no distinct mass is palpable in it. It is covered by a dressing applied to the site injected by Mr. Braund. In the left axilla there are enlarged glands; on the left side of the neck there are numerous small, hard, enlarged glands extending upwards into the posterior triangle of the neck for three or four inches. There are also enlarged cervical glands in the right posterior triangle of the neck. X ray shows metastatic deposits throughout both lungs with increased opacity over right diaphragm, suggesting either fluid or consolidation. Hilar and mediastinal glands also enlarged.

Comment.—This patient is not cured. She has widespread cancer and her prospects are very grave. She has received no benefit from the treatment.

CASE C.—Male; age 38 years. Cancer of the tonsil. Growth in left tonsillar area removed on 15.10.46. Biopsy showed it to be anaplastic carcinoma of tonsil. Four courses of X radiotherapy over twelve months. During this period recurrence occurred in tonsillar region with extension to the tongue and a large nodal mass became palpable in left submandibular region. Consulted Mr. Braund on 10.1.48, when he was given an injection in the cheek. The injection caused great pain over whole left side of face for about six hours. The injection was not made into the fungating tumour in centre of cheek, but between the tumour and the ear. The site of injection became black within twenty-four hours and extended over an area about two inches square. Within fourteen days the black portion commenced to slough from tissues and on the seventeenth day Mr. Braund removed "a lump of tissue about the size of a match box", leaving behind what Mr. Braund termed "three roots which would come out". For a time after this his appetite improved and he put on weight, but has lost about a stone recently.

On 15.3.48 Mr. Braund gave the patient another injection in the left side of the neck below the angle of the jaw. This caused pain of a stinging nature which lasted about one hour and the part injected became black within twenty-four hours.

Patient was examined on 19.3.48, when he was in good general condition but sallow. Left side of the face is occupied by a large hard swelling from the central portion of which a fleshy mass of growth is fungating. His mouth is tightly closed; unable to open it. The swelling of the face extends from the angle of the jaw to within one inch of the mouth and at the posterior portion of it there is a healed scar which was the site of the first injection. Over the upper portion of the sternomastoid muscle at the site of the second injection there is a black slough triangular in shape with base above one and a quarter inches in length and the two sides one and a half inches in length. Below this slough there are some small palpable cervical glands. It is difficult to say whether these are inflammatory or neoplastic. Through a gap in the teeth a fungating ulceration of the left cheek can be seen extending towards the tonsil.

X-ray films taken 19.3.48 show: A retained dental root in the left side of the mandible in the canine region. There is some infection around this root. There is no evidence of any carcinomatous invasion of this bone. Film of thorax shows lung fields clear. Costophrenic angles normal. Apices clear. Heart and great vessels within normal limits.

Comment.—This is a rare type of cancer. It is not cured, but has in fact extended under Mr. Braund's treatment. It must be remembered that for fifteen months before seeing Mr. Braund he had been under medical radiotherapy and there is no evidence that it has spread to internal organs. Though not a rapidly fatal case of cancer, the eventual outlook is grave.

CASE D.—Male; age 53 years. In February, 1943, had a sore on margin of lower lip on left side, which was treated by X rays. The diagnosis at this stage is uncertain. He consulted another doctor in October, 1944, complaining of a lump in his neck which had been present for two weeks. This lump was considered to be a secondary glandular metastasis attached to the mandible. X ray was negative. On 27.10.44 submandibular dissection and removal of the mass with the outer table of the mandible was carried out. *Unfortunately, no histopathological examination of the mass was made.* X radiotherapy was given. Patient states that he was well until November, 1947, when a spot appeared in the centre of operation site the size of a pin's head. This formed an ulcer and gradually extended. He sought treatment on 10.1.48 from Mr. Braund, who has applied a fluid about once each fortnight. This causes pain for about two to two and a half hours. He has not been treated by injection. Examination on 12.3.48 showed in the left sub-

mandibular region an ulcer about two inches in diameter exposing the jaw, the exposed bone being necrotic and discharging pus. The margins of the ulcer are indurated, especially in the submental region. There was no abnormality in the mouth. The alveolus was normal and there was no evidence of involvement of the mucous membrane on the floor of the mouth or of the cheek. No enlarged glands were detected. X-ray films showed osteomyelitis of the middle third of the left side of the mandible with a large sequestrum which has probably separated. Radiographically there is no evidence of carcinomatous infiltration of the bone.

Comment.—The diagnosis in this case is uncertain; that being so, we do not see how Mr. Braund can know that this is a case of cancer. This can only be determined by a microscopic examination of some tissue surgically removed or by the eventual outcome which we cannot wait for.

CASE E.—Male; age 51 years. Developed a patch of leucoplakia on lower lip about fifteen years ago which was treated with diathermy. Lip tended to split and become inflamed for about ten years when it was treated with radium. Subsequently a lump formed just below margin of lip which was removed by v-shaped incision. About three to four months later glands developed in the neck on each side and also in middle line which were operated upon in October, 1946. In October, 1947, he noticed a lump in the left anterior triangle for which further operation was advised.

At this stage he sought treatment from Mr. Braund, who gave the usual preliminary hydrotherapy and massage and injected the lump on 30.12.47. The slough commenced to separate about a fortnight later. The site of injection was still discharging slightly on 5.3.48.

Examination of the patient by the Committee took place on 5.3.48, when the following findings were recorded:

There has been extensive dissection on the right side of the neck including the removal of the sterno-mastoid muscle. On the left side only the upper cervical region has been dissected and the sterno-mastoid muscle is still present. Over the upper end of the sterno-mastoid muscle on the left side there is a plaster dressing about one and a quarter inches in diameter covering the site injected by Mr. Braund. Beneath the dressing a small tumour is palpable and above it immediately below the angle of the jaw another nodule can be felt with an approximate diameter of about half an inch. No other glands can be detected and there is no evidence of recurrence on the lip. X ray shows no evidence of chest involvement.

Comment.—Although the Committee does not possess the essential proof of histopathological examination of the patient's tissues, they are satisfied that the condition is due to cancer (epithelioma) probably of low degree of malignancy.

This patient is of special interest in that he was one of several who appeared in a Cinesound film shown on 5th February, 1948, when he proclaimed that he had been cured of cancer by Mr. Braund in a few weeks.

The Committee is definitely of the opinion that such optimism is contrary to fact.

CASE F.—Female; age 44 years. Carcinoma of the right breast. Radical removal of right breast 15.7.42. Discharged from hospital 5.9.42 relieved. Readmitted 9.4.43 for malignant glands in neck which were removed surgically with uneventful recovery, but bad prognosis.

Patient commenced treatment with Mr. Braund about mid-December, 1947. Treatment consisted of hydrotherapy and massage. No injection was given by Mr. Braund. Examination on 12.3.48 showed numerous secondary deposits of carcinoma in the skin medial to the scar and extending into the left breast where there is a large irregular mass in the inner and lower quadrant associated with "pig-skin" appearance of the lower half of the breast. Secondary deposits above the right clavicle, one being fixed to the outer end of the clavicle, exceedingly tender and more than an inch in diameter. There are secondary deposits extending from the right axilla downwards into the arm for two or three inches. Glands are palpable in left axilla and above the left clavicle.

Comment.—This is a case of proved cancer where the patient has survived for nearly six years prior to seeing Mr. Braund. It demonstrates clearly that cancer is not invariably a rapidly fatal disease and shows the futility of Mr. Braund's claim that his treatment, when not curative, will prolong life. It is obvious that he has not cured her and there is no evidence that he has prolonged her life. The outlook is grave.

The following patients were not seen by the Committee; but as their histories are available they are cited.

CASE G.—Female; age not ascertained. Carcinoma of the right breast. First consulted her doctor in July, 1947, when he found a mass in the right breast infiltrating into the anterior axillary fold. X radiotherapy brought about almost complete disappearance of the mass and also disappearance of the puckering of the axillary fold. Radical operation in August for removal of breast and resection of axilla.

Several weeks after returning home she complained of a dull ache in the lower back with vomiting and nausea. X-ray examination revealed multiple deposits in second, third, fourth, fifth lumbar vertebræ, sacrum, both ilia, both pubes and the superior half of both femora.

About this time the patient commenced treatment by Mr. Braund, but the actual treatment given is not known to the Committee. Arrangements were being made for the examination of the patient when, unfortunately, she died on 5.3.48, the certified cause of death being: Metastatic carcinoma following carcinoma of right breast.

CASE H.—Female; age 37 years. Anaplastic carcinoma of the left breast. First consulted a doctor on 30.10.47, when she had a large mass in the left breast about seven inches in diameter fungating in one area, hard, with "pig-skin" appearance in another area. Definite axillary glands were palpable. X radiotherapy administered before any consideration was given to surgical treatment.

Patient sought treatment by Mr. Braund, who presented her to a member of the Committee (who was making preliminary inquiries in an official capacity into Mr. Braund's claims) on 9.1.48. Mr. Braund stated that she was an undoubted case of cancer that he undertook to cure.

The breast had been injected by Mr. Braund some time previously, the injection having been given in the lower inner quadrant. There was a suppurating gangrenous area about three inches in diameter extending deeply into the breast tissue. Glands were palpable in the axilla. The whole breast was congested and the patient looked very ill, though she stated that she was much better and was being cured.

The patient resolutely refused to be examined by the Committee, but consented to the doctor, under whose care she now is, issuing a report. The report, dated 15.3.48, reads: ". . . has a large triangular sloughy ulcer of her left breast (4 by 5 by 5 inches) with raised hard edges about one inch wide. The breast itself is œdematous. There are large glands of rubbery consistence in both axillæ and there is also a small hard nodule in the other breast. I cannot detect evidence of metastases clinically elsewhere. She is confined to bed and her general condition has markedly deteriorated since I first saw her on 27.2.48. In my opinion her expectation of life is not long."

Comment.—This patient, obviously ill, appeared in the Cinesound film where she stated in effect that Mr. Braund had "removed a cancer from her breast without bleeding, without anæsthetic, without a knife, and without pain. No one else could do that."

CASE I.—Female; age 72 years. Carcinoma of the œsophagus. About August, 1947, the diagnosis of carcinoma of the postcricoid region of the œsophagus was made after X-ray and œsophagoscopy examination. Biopsy confirmed the diagnosis of squamous carcinoma.

It was considered that X radiotherapy would be of no value and the patient then placed herself under Mr. Braund. She was apparently treated by hydrotherapy, etc., for a long period and eventually given three injections in the suprasternal region, but Mr. Braund "could not draw out the cancer". Mr. Braund then gave an injection into the back of the neck and, it is alleged, subsequently told her that her neck was dislocated. He "reduced the dislocation by manipulation", but as this was a very painful process and as he could not cure her the patient ceased to attend Mr. Braund.

The patient again came under medical care on 26.2.48, when she could only swallow fluids, and palliative gastrostomy was done on 9.3.48. She has now a gastric fistula for feeding purposes and can only swallow liquid with difficulty. Her expectation of life is very limited.

Comment.—This is a case of cancer that Mr. Braund was actually the first to treat. She was not treated by surgery nor X radiotherapy which Mr. Braund contends, quite erroneously, interfere with the efficacy of his treatment.

Publicity.

The publicity given to Mr. Braund's claims in a section of the Press, in the Cinesound film "Can Braund Cure Cancer?" and through certain other channels is regrettable.

Claims made in the Press were in most instances extravagant and unwarranted, and having been transmitted as "news" to all parts of the world were given an international importance which was totally undeserved.

Telegrams and air mail inquiries were received from the United Kingdom, several European countries, the U.S.A., Canada and even from Iraq.

Several persons travelled to Australia by air from Canada to obtain treatment, and undoubtedly false hopes have been raised in thousands of sufferers throughout the world.

At its first meeting the Committee became aware that a film relating to Mr. Braund's treatment was about to be released. The Committee resolved:

> That the Committee recommend to the Government that appropriate action be taken immediately to ban for the present the public exhibition of films taken on the alleged removal of cancer by Mr. John Braund from the bodies of patients, and that such ban continue until such time as the Committee is satisfied that it was in fact a successful removal of cancer.

The Committee was informed later that "the Government has no authority under which it could prohibit the public exhibition of this film".

Meanwhile the film was released by Cinesound, but after some time was withdrawn.

The film can only be regarded as misleading propaganda for Mr. Braund with a final saving statement that the motive is to bring the question of his claim before the public. Out of five patients shown in the film as having been cured of cancer, two never suffered from that disease, two are mentioned in the case histories (Case E, epithelioma of lip with gland involvement, and Case H, cancer of breast. Expectation of life very short) and one, who was too ill for the Committee to examine, is still suffering from the disease.

It was intended to have the film released in several countries overseas. The Committee feels thankful, for the sake of Australia's good name, that this proposal has apparently been abandoned.

Mr. Braund's Secret "Cure".

Up to this point the report is as submitted for adoption by the Committee.

During the discussion on the motion for its adoption, the Chairman (Dr. E. S. Morris) stated, at a particular stage, that he considered it necessary to make an important statement to the Committee. His statement, which follows, is incorporated in this report, together with explanatory comments by Dr. Brose, at the direction of the Committee.

CHAIRMAN: I think at this stage I ought to make a very important statement to this Committee. I do so under a deep sense of the responsibility and with a full appreciation of the gravity of the remarks which I am about to make. I desire to emphasize at the outset that I have not consulted a single member of this Committee in regard to the proposed action that I am taking. That course was adopted for two reasons: First, that above all things I wanted to satisfy myself finally as to the truth. I was not able to do that until the last few days. The second reason was that I did not wish to implicate any of my medical colleagues in this matter and leave them or myself open to an appeal to popular prejudice that this was a conspiracy on the part of the medical profession to suppress the "world-shaking discovery" of Mr. Braund. Dr. Brose's opposition to this report is not unexpected. I should have been rather surprised if he had been prepared to agree with it. In one section of the report attention is directed to the striking similarity between the Koch-Baker treatment and Mr. Braund's treatment. The connecting link between those two treatments is Dr. Brose, who has been closely associated with both of them.

DR. BROSE: In what way do you mean "closely associated"?

CHAIRMAN: You were associated with the Koch-Baker treatment in New Zealand, and you have been closely associated with Mr. Braund. But you can have your say later. I find it difficult to believe that that connection or association is purely fortuitous or accidental. Dr. Brose has stated publicly, in the Press and otherwise, that he has been closely associated with Mr. Braund for some four years, during which period he has carefully watched his treatment of patients, and has formed the opinion that his treatment possesses considerable merit. Dr. Brose appeared in the Cinesound film entitled "Can Braund Cure Cancer?" in support of Mr. Braund, and as an alleged cancer expert, implied, or allowed the implication to be drawn, that Mr. Braund's treatment has scientific support, or at least was worthy of scientific support.

DR. BROSE: What do you mean by "scientific support"?

CHAIRMAN: You came out as a scientist in the film. That is the whole thing that gave the film any weight at all, and you claimed to be a scientific cancer expert.

DR. BROSE: Not a scientific cancer expert; a cancer research worker.

CHAIRMAN: Let me have my say now; you can say what you like afterwards. Dr. Brose has informed this Committee definitely and personally that he strongly supports Mr. Braund's treatment, and he certainly has given us definite information which allows us to form the opinion that he is guide, philosopher and friend to Mr. Braund. Dr. Brose has admitted that whilst a member of this Committee he wrote a letter, dated 14th March, signed by Braund and forwarded to the Premier, in which allegations were made, *inter alia*, that the Committee's investigations were unfair. The import of that letter, as you know, appeared in the Press the day following, long before this Committee could be called together to consider it. The allegations were proved at this table to be incorrect, and you, Dr. Brose, admitted that you were satisfied on that point. You were in a position to ascertain the true facts long before you wrote that letter had you wished to do so, and you did not. Is it any wonder that one member of the Committee at the meeting that considered that letter stated to you, in front of all of us: "I wish Dr. Brose would cease running with the hare and hunting with the hounds." Do you remember that?

DR. BROSE: Dr. Morris, my whole interest in this matter is purely impersonal.

CHAIRMAN: You can have your say afterwards. I mention these facts to emphasize the intimate personal relationship that exists between Dr. Brose and Mr. Braund and to say definitely that Dr. Brose is an open and avowed advocate for Mr. Braund's treatment. I think that is a fair statement. Is it unfair, in the circumstances, to infer that Dr. Brose probably knows the intimate details of Mr. Braund's treatment, and is probably quite aware of what Mr. Braund injects into his patients?

DR. BROSE: I have not the faintest idea.

CHAIRMAN: I will not ask you whether you know, because you would probably tell me I had no right to ask the question, and you would not answer. But I make two assumptions—the first, that you do not know, and the second, that you do know. Dealing with the first—that you do not know—in that case this Committee is asked to believe that you, a scientific man, with scientific training and background, and no doubt with a fairly acute mind—as everybody around this table recognizes—claiming to be a cancer expert associated with Mr. Braund for some four years in the active treatment of his patients and with ample opportunity to discover any fact that you wanted to for yourself, have been hoodwinked by a comparatively ignorant man like Mr. Braund. That is a picture that my imagination cannot form. I am not able to visualize you as an innocent babe in the woods. It ascribes too much credulity to you and too much mental acumen to Mr. Braund. As members of this Committee have seen both you gentlemen, however, I will leave it to their individual judgement to form their own opinions.

Let us take the second assumption—that you do know. If that is accepted, then this Committee can only reach one conclusion—that you have been an active partner with Mr. Braund in his activities and in the treatment of his patients, and if I can prove—as I hope to prove presently—that Mr. Braund's treatment is a nefarious fraud, then you are inextricably bound up with him in what amounts to causing an outstanding public mischief. Those are strong words, and they are intentional.

DR. BROSE: Well, Dr. Morris——

CHAIRMAN: Just a moment, please; you can have your say afterwards. You will remember, gentlemen, that we attended Mr. Braund's house on the 1st March. That is a very important date. In detective stories, as you know, what appears to be a perfect crime is frequently unravelled by some slip of the criminal. On that date you will remember that Mr. Braund committed a very slight mistake. He gave me a piece of tissue, which you saw taken out of the breast of Mrs. A.G. I have already told you that I brought that back and it was given to Dr. Morgan first to take a section of it. Dr. Morgan found, on microscopic examination, what everybody thought would be found, that it was merely necrotic tissue. As a stab in the dark—a million to one chance—I felt that it would be necessary to take another step. I did so because I thought that it was not fair to leave any opening unexplored, and on that account I gave the rest of the tissue to Dr. H. B. Taylor, the Government Analyst, and asked him, if he could, to ascertain any basic metals or other substances that could

be detected in the tissue. I was quite surprised with the result.

I shall read you Dr. Taylor's findings presently, but here let me say, for the benefit of the Committee and for the benefit of Dr. Brose, if he says he does not know, the great and wonderful secret cure of Mr. Braund is the injection of ordinary commercial alum into the tissues of the patient. I should now like to read you Dr. Taylor's "Report on a specimen of tissue received from Dr. E. S. Morris, Director-General of Public Health, on 8th March, 1948".

(Reads report) . . .

"I, the undersigned, am the Government Analyst in the State of New South Wales.

"My qualifications are: Doctor of Science of the University of Sydney; Fellow of the Royal Institute of Chemistry of Great Britain and Ireland; Fellow of the Australian Chemical Institute.

"My experience in the chemical analysis of biological material embraces the determination of trace metals and the complete analysis of the inorganic constituents of a large number of miners' lungs. This work was published in the Annual Report of the Department of Public Health, N.S.W., 1936.

"I hereby certify that I examined a specimen of tissue received from Dr. E. S. Morris, Director-General of Public Health, with the following results:

> "The specimen weighed 2·0 grams, it had an ash of 0·012 grams and was found to contain 2·5 milligrams of aluminium and 1·6 milligrams of sulphate. These figures, calculated to 1 gram of ash, become 210 milligrams and 140 milligrams respectively.

"In the following table a comparison is made between the aluminium and sulphate content of the specimen and of other tissues:

Sample.	Weight. (Grams.)	Ash. (Grams.)	Milligrams per Gram of Ash.	
			Aluminium.	Sulphate.
1. Specimen ..	2·0	0·012	210	140
2. Liver tissue ..	100	1·14	0·1	13
3. Breast tissue ..	141	0·16	0·1	14
4. Cancer tissue (breast) ..	67	0·023	0·1	—
5. Guinea-pig ..	0·7	0·012	340	—

"From the above table it is clear that the specimen marked (1) in the table contains both aluminium and sulphate radicals largely in excess of specimens of normal tissue marked (2) and (3) and also of a cancerous tissue marked (4)."

We are extremely fortunate in having not only such an able chemist as Dr. Taylor, but also in having a chemist who has had unique experience in the analysis of human tissues. To check up on the possibility that it could have been an artefact, Dr. Taylor examined liver tissue, which, as medical men will know, would contain any aluminium or anything else absorbed from the intestines, and he took a very large quantity of liver and on analysis it had less than 0·1 milligrams per gram of ash compared with 210 milligrams in the specimen of tissue I referred to. Then we took breast tissue and that came also to less than 0·1. Cancer tissue of the breast which we also examined had less than 0·1. Then a guinea-pig, which we will deal with later, was treated. The slough from the guinea-pig was examined and it contained 340 milligrams of aluminium. I will come back to the guinea-pig presently. What I want you to know at the present time is that the amount of aluminium found in that slough taken from the breast of Mrs. A.G. contained more than 2000 times the amount of aluminium found in normal human tissue.

"In order to obtain information on the effect of aluminium sulphate on tissue, one rabbit and two guinea-pigs were injected with autoclaved solutions of potassium aluminium sulphate (alum) and aluminium sulphate."

That last is the pure chemical. This is what happened:

"*Rabbit*—

Right leg injected with 1 ml. 20 per cent. potassium aluminium sulphate on 18th March, 1948. Within forty-eight hours the area of injection had turned black.

On the sixth day the rabbit did not eat.

On the seventh day it had recovered.

On the thirteenth day (31st March) a black scab had commenced to separate with a well-defined area of slough underneath.

Left leg injected with 1 ml. 20 per cent. aluminium sulphate."

That is the pure chemical again.

"On tenth day area of injection was only slightly discoloured. No scab formation.

On thirteenth day same appearance as for tenth day.

Guinea-Pig No. 1—

Injected on right leg with 0·5 ml. 20 per cent. potassium aluminium sulphate. Within forty-eight hours the area of injection had turned black."

I want you to note specially that turning black and I will direct your attention to that later on.

"On sixth day it did not eat.

On thirteenth day a black scab with a well-defined slough area had commenced to separate.

The guinea-pig was killed and held for inspection.

Guinea-Pig No. 2—

Injected on right leg with 1 ml. 10 per cent. aluminium sulphate.

On sixth day it did not eat.

No darkening of injected area before tenth day.

On twelfth day black scab had formed with well-defined slough area.

The guinea-pig was killed for inspection and the slough analysed (marked (5) in table)."

The slough was taken out just like Mr. Braund takes out his "cancers" and subjected to an analysis to show what would be the chemical findings on such a slough. That guinea-pig showed, as I told you, 340 mg. per gram of ash.

"From animal experiments it is seen that after injection with potassium aluminium sulphate a black area is formed at the site of injection in from twenty-four to forty-eight hours, and this black area rapidly forms into a scab with a well-defined slough area commencing to separate on the thirteenth day.

"After injection with aluminium sulphate (pure) little obvious change takes place for ten days.

"I have been informed that the specimen of tissue submitted for analysis had been removed from a patient at the site where an injection of some substance had taken place and also that the area of injection had turned black in about twenty-four hours.

"In view of this information and the facts disclosed by the analysis and the animal experiments, (1) that the specimen contained relatively large amounts of aluminium and sulphate, and (2) that after the injection of potassium aluminium sulphate the site of injection turned black within forty-eight hours, I am of the opinion that the substance injected was alum.

(Sgd.) H. B. TAYLOR,
Government Analyst."

I want to stress here what I have told you already, that the specimen contained more than 2000 times the amount of aluminium found in normal human tissues and approximately ten times the amount of sulphate. I drew your attention to the fact that the area went black. If you refer to case history C you will find that the patient gave me a history that within twenty-four hours after injection by Mr. Braund the area went black where the substance had been injected. When we saw the patient some days later at Sydney Hospital the area was black and necrosing. Now the connection or the similarity—the necessary consequence if you like—of the injection of alum with what actually occurs in Mr. Braund's patients I leave to your own individual judgement. The analysis of cancer tissues was done in order to avoid any fantastic view that may have been put forward that cancer tissue absorbs all the aluminium that is available or possible to be utilized in the human body and that the aluminium comes out with the slough when it is removed from the body. To avoid that possible fantastic theory we analysed the cancer tissue and you will see that cancer tissue has exactly the same mere trace of aluminium as any other part of the human body.

I have Dr. Taylor available, and if any member of the Committee would like to question him I will bring him in. He is available if any member of the Committee wishes to see him and ask him any questions. What is your wish?

MR. HALLSTROM: I think his evidence would be very important.

(Upon the arrival of Dr. H. B. Taylor, Government Analyst, members of the Committee examined the guinea-pigs referred to in the report.)

CHAIRMAN: Dr. Taylor is now available for questioning, gentlemen.

DR. COLLINS: Suppose that along with the large amount of alum that was injected into the patient there were also some other substances—other drugs—that we might call,

for purposes of argument, "X" and "Y". What would be the fate of "X" and "Y" at the site of injection?

DR. TAYLOR: I would think that the alum having carried out its work of coagulation, would block entry into the body of any other substances mixed with it.

DR. BROSE: Following upon that, may I ask the time of coagulation before it takes effect? Is it instantaneous or would there be time for some other substance to get in, even in small amounts?

DR. TAYLOR: I could not give an actual time factor, but I would say it would be very short—five or ten minutes and no more.

DR. BROSE: In that time some other substance could get into the circulation? Let us say that there was some ammonia mixed with it. That would get in at the same time also?

DR. TAYLOR: Yes, but it would be in very small quantities indeed.

DR. BROSE: If this is what Mr. Braund uses—and I do not know what it is—he gives it in very small quantities of no more than a couple of minims.

DR. COLLINS: What effect would the buffering the blood stream have upon any ammonium that reached it? It would be at once neutralized?

DR. TAYLOR: I think that it would.

DR. NISBET: Could you assess by the amount of aluminium you found in the tissues in the Braund specimen the amount of fluid injected?

DR. TAYLOR: No, that would depend entirely upon the strength of the solution injected. From the last number, number five, in the table of the report I see that the amount of aluminium found was 340 milligrams. That is the quantity per gram of ash, and the reason for multiplying it is to make all the figures comparable, as the amounts of ash vary. I would think that this 0·7 weight of the guinea-pig, as compared with the two grams of the specimen—the two ashes being the same—the injection of one c.c. (or one ml.) of a 10 per cent. solution, would possibly be somewhat in excess of the amount given to produce 210. In other words, those two figures after all are of the same order and, to go to extremes, I would not expect the amount injected in the guinea-pig to be ten times the amount injected in the patient. One cannot go down to small variations, but I would say it is of the same order, and I would imagine that something like a c.c. of a 10 per cent. solution would be somewhat about the amount given.

DR. BROSE: What would a 10 per cent. solution look like?

DR. TAYLOR: It is quite clear; it is quite soluble.

DR. BROSE: The solution injected by Mr. Braund is definitely milky in appearance, like a colloidal solution. I was wondering if he had it very strong, whether it would clear, or not dissolved?

DR. TAYLOR: If you autoclave solutions of either aluminium sulphate or of potassium aluminium sulphate a deposit will occur and turn the solution milky. If I take a 10 per cent. solution of potash alum and autoclave it to make it sterile, then that solution would become milky.

DR. TEBBUTT: Would that happen if you boiled it?

DR. TAYLOR: I imagine so; I did not boil it. It is simply a deposit of hydrated aluminium oxide.

DR. TEBBUTT: I cannot remember the formula for ammonia alum. Is there any sulphur in that?

DR. TAYLOR: It is just a substitution of two ammonias for two potash. It is ammonia aluminium sulphate.

DR. BROSE: That would explain the ammonia. Ammonium alum could be used instead of potash alum?

DR. TAYLOR: There is one interesting point here that I have not mentioned in the report. In 4 and 5 of that table I have given no sulphate figures because if aluminium sulphate is heated to a dull redness approximately 500 degrees Centigrade, it will start to lose sulphate. In other words it will start to decompose and at 750 degrees has completely decomposed. For the pig I used for injection aluminium sulphate, and therefore in my ash I lost my sulphate because I ashed at between 500 and 600 degrees. In the specimen I did not lose all the sulphate. If potassium aluminium sulphate is heated, only 75 per cent. of the sulphate is lost—25 per cent. remains combined with the potash. If it had been ammonium alum, ammonium sulphate is volatile and I think I would have lost all the sulphate and aluminium only would have remained. It is only an assumption, but I think that it bears out the evidence given from the injections, that a stable form of alum was used rather than either aluminium sulphate or ammonium.

DR. BROSE: Suppose that there is a mixture. Would that explain it? I want to find out why there was a smell of ammonia. There might have been a mixture of three alums?

DR. TAYLOR: It would be difficult to trace the ammonia. I could not trace the ammonia, nor could I definitely say that it contained potash or soda because my tissues contained potash and soda. Unless I had some sort of comparison as a control I would have to conclude that my potash and soda were derived from body tissue. That is the only thing that I am caught on.

CHAIRMAN: The important point is that there is no doubt in your mind, as proved by the analysis, that the amount of aluminium in that tissue must have been introduced into it?

DR. TAYLOR: I am quite certain of that.

DR. BROSE: You feel certain that it was one of the constituents?

DR. TAYLOR: As I have put it here, I feel certain that both aluminium and sulphate radicals were present. I put it that way rather than say it is an aluminium sulphate—which I cannot say. Both of these radicals have been introduced, but I am unable to say whether other radicals have been introduced in addition.

DR. BROSE: You could not be sure of potassium?

DR. TAYLOR: I am only working with 12 mg., which is a very small amount.

DR. BROSE: Therefore you could not detect any change in the blood or something that had been introduced there. The blood volume would be far too small?

DR. TAYLOR: No, I went through the whole gamut of my heavy metals before I got to aluminium and the total volume I had to work with was something like 2 c.cs.

DR. BROSE: Micrometrical methods were used?

DR. TAYLOR: It was minute work.

CHAIRMAN: Another important point is that it would be the aluminium that produces the slough rather than anything else?

DR. TAYLOR: Yes.

CHAIRMAN: You are quite certain on that point?

DR. TAYLOR: I have not injected aluminium oxide into guinea-pigs.

CHAIRMAN: It is the aluminium—not the sulphate or ammonia or potassium?

DR. TAYLOR: I would say that it is aluminium, because that is the astringent agent which does the sealing.

CHAIRMAN: Have you gentlemen any other questions to ask of Dr. Taylor?

DR. BROSE: There is no way of working out what proportion of the injection could have been aluminium, or alum, or some salt of aluminium?

DR. TAYLOR: Yes, there is one way. If you gave me a dozen sloughs I could give you the figures. I could compare them with the normal content.

DR. BROSE: There is no other radical that you feel might have been there—silicate or something of the sort? Were other radicals tested for?

DR. TAYLOR: A certain amount of silica was present, but silica was also present in all my other tissues. The radical that I have given—aluminium—was the abnormal one as compared with the controls and was the only abnormal one. The iron was there, calcium was there as phosphate and the alkalis, but the aluminium was the only one present which was not present in the other samples.

DR. BROSE: Did you test for antimony? I have heard it said that it can also cause sloughs. Did you test for zinc?

MR. HALLSTROM: Would zinc chloride have the same effect?

DR. TAYLOR: I imagine so, but zinc was not present. These are the metals that I started with: silver, zinc, iron, gold, cerium, selenium, tungsten, molybdenum, silicon, lead, bismuth and antimony. They are all negative with the exception of iron and silica.

DR. BROSE: There was only a trace of silica?

DR. TAYLOR: Yes, it was there in all cases.

DR. BROSE: The silica would be abnormal?

DR. TAYLOR: No, it was in all my other tissues. The only abnormal metal was the aluminium.

(Dr. Taylor at this stage retired.)

CHAIRMAN: To resume, I only wish to say, like the lawyers, that I now rest my case. I hope that I have proved to the satisfaction of everyone, right up to the hilt, unless Dr. Brose can give any satisfactory contrary explanation, that Mr. Braund is a fraud and a charlatan and that what he is using is alum; further, that it is a delusion and a snare. The implications of that is a matter for your judgement, and I think that we should hear anything that Dr. Brose would like to say in regard to my statement or the evidence that I have put forward.

DR. BROSE: It is rather a long story and I have to think back. Perhaps I should start at the latter end first. So far as the material injected is concerned, it appears certain that alum in some form or another—there are several alums—has been used. From what I saw on the day that I was at Mr. Braund's place and he was giving injections that was not

the only element in it, though it may have been an important element. I do not know the composition of the material used, any more than I know what the Baker material was.

CHAIRMAN: You must be very trusting.

DR. BROSE: It was not my business to find out what it was.

CHAIRMAN: Even though you were advocating something?

DR. BROSE: Not to find out what was being injected. I was interested in the results on certain patients. That alone justified an inquiry. I did not know Mr. Braund very well personally and I was there simply as an interested person. I have always been interested in that sort of work, and I felt that he was getting certain results that should be investigated. I still think that he has been judged too harshly. You have only to consider Mr. R. and others to know that. After all, there were very few cases examined, and you gentlemen will agree with me on that point, to form a judgement. So far as the actual material is concerned, I do not know what it is, neither do I profess to know. I did not expect to know what it was until it became public. People have written to me from several places. An old pastoralist came to me and spoke of a substance that also produced a slough. He had had some success in using it with cattle. There were one or two other things that he said about it that made me feel that it was not the same substance. He told me that it was a mixture of three things. One other gentleman spoke of a certain juice. Lots of people hear of things like this pastoralist. However, I found that the substance was not unknown to trainers of horses, who have a similar treatment, which is probably the same. I am sorry that the New Zealand matter was brought up because it has no association with this inquiry and is irrelevant to it. We were asked to deal with Braund and not with New Zealand or with Roberts.

DR. EDYE: You were in New Zealand with Dr. Williams and were working with him there?

DR. BROSE: I was there.

DR. EDYE: You were interested and you were helping him?

DR. BROSE: I was not helping him. I would now like to read, for the information of you gentlemen, a letter that came to me from the office of the Minister of Health, Wellington, from Mr. Frazer, who is now Prime Minister. (*Reads letter.*)

DR. BROSE: The feeling mentioned in that letter was not directed towards me. About two months later the writer sent me a present of a book and wrote in very cordial terms. The feeling was between himself and the other person.

DR. EDYE: Have you the original letter?

DR. BROSE: Yes.

DR. EDYE: I think it ought to be presented.

DR. BROSE: I will not present it, but I will lend it.

CHAIRMAN: May we have the copy, and also the copy of your original letter?

DR. BROSE: Here is the copy of the original letter. (*Handed to Chairman.*)

DR. EDYE: Who was Mr. McCulloch?

DR. BROSE: He was the man in Queensland.

CHAIRMAN: He had an institute of physiotherapy.

DR. EDYE: Was he qualified?

DR. BROSE: No.

DR. EDYE: Was he the man who used arsenic?

DR. BROSE: I do not know what he used.

DR. EDYE: What did he call himself?

CHAIRMAN: He called himself a naturopath.

DR. EDYE: Were you working with him?

DR. BROSE: No. I was not working with him in any way.

DR. EDYE: But your name is associated with his in that letter.

DR. BROSE: That has nothing to do with working with him. You might get an expert to come along and require help from him in some matter, but that is not working with him. If the Committee intends to include the Koch-Baker business in its report I think it ought to add this letter that I wrote to Sir James Elliot. I consider that it is all irrelevant, but if the thing is to be done at all it ought to be done thoroughly.

CHAIRMAN: I do not know that this letter is relevant to the matter at all.

DR. BROSE: It is just as relevant as bringing the Koch-Baker treatment into the report. You, Mr. Chairman, made definite reference to that.

CHAIRMAN: I referred to it intentionally because I think it is very significant. It is for this Committee to judge that you were closely associated with both the Koch-Baker and Braund treatments. That is the implication, and you are well aware of what it means.

DR. BROSE: Am I to take it that you think that I know what was in the Baker and Braund treatments, and that I have tried to work the two together, or that I am interested in any way? If it is so, I would like you to say so.

CHAIRMAN: I am leaving it to the open judgement of the Committee.

DR. BROSE: It is not a matter of leaving it to judgement. If you suspect me of that, I should not have been on this Committee.

CHAIRMAN: I think it was most improper, if not immoral, that you should be on this Committee as an open advocate of Mr. Braund. I do not think, if I had been in your place, I would have gone on the Committee.

DR. BROSE: I advocated an open investigation. That is what I felt should be done. There are possibly other methods of a similar nature which should be tried out. Why should the Committee be afraid of trying out something?

DR. EDYE: I take a point of order. We cannot go on like this all day. It is wasting our time.

CHAIRMAN: I agree. I have made a straight-out presentation of a case which implicates you with Mr. Braund. You have had a fair opportunity of hearing all the evidence and cross-questioning Dr. Taylor. If there is anything you want to say in explanation, by all means say it; but I do not want to go over the whole ground.

DR. BROSE: I am not implicated with Mr. Braund in any way. My interest is in any cancer cure that I think holds out a certain possibility. I saw certain results that encouraged me to think that Mr. Braund's methods should be investigated. But that is my interest in the whole matter. I have never helped him to treat a patient, and I do not know him in his private capacity. I have gone out there, as others from the Committee have gone out, and I have been interested.

CHAIRMAN: That is not the impression that is conveyed to the public.

DR. EDYE: You wrote one letter.

DR. BROSE: He told me those were his ideas, and I put them into words for him. I think it is a pity I did, because it has given a misconception.

DR. EDYE: You have met Mr. Braund socially. You admitted it at the last meeting.

DR. BROSE: That was done with a purpose—to make contact with him.

DR. COLLINS: Did you not contribute an article to *The Sun*, praising Mr. Braund's methods?

DR. BROSE: Saying I thought there was something in it which should be investigated.

DR. COLLINS: You gave him a nice plausible theory.

DR. BROSE: That theory was what I was convinced was happening. Even with that analysis I think you heard Dr. Taylor say it would take five to ten minutes for coagulation to take place. If he is injecting some other substance, it would have time to get into circulation. That is a possibility.

DR. EDYE: Dr. Brose has admitted that he was an advocate of Mr. Braund all along, and we have sufficient proof from the article in *The Sun*, which openly shows that he was an advocate of Mr. Braund. We do not want to investigate it any more. We know he was an advocate of Mr. Braund's and he admitted it in this meeting, and he wrote his letters. If that is not sufficient to satisfy anybody, I do not know what would be.

DR. COLLINS: I should like to move at this stage, Mr. Chairman, that the remarks that you have made latterly in this meeting, and the reply that Dr. Brose has made, should be included in, or appended to the report. That would include all the information we have obtained from Dr. Taylor. I should like an opportunity to speak later, but for the purpose of this motion I do not wish to add any discussion.

DR. TEBBUTT: I second the motion.

CHAIRMAN: The motion is, "that my statement to the Committee, including Dr. Taylor's certificate, together with Dr. Brose's reply, should be added to the report". Is there anything more you would like to add before we put the motion?

MR. TREATT: I would like to ask Dr. Brose whether he would tell us what he thinks, in the light of Dr. Taylor's evidence on the analysis. He heard the evidence. As a scientist, what does he think of the treatment now?

DR. BROSE: I do not really think I am competent to say that because, although I have seen the result of the guinea-pig experiments, what really matters is whether the stuff that is injected—alum or partly alum—produces the result that Mr. Braund claims from it. It might have been some complex organic substance, but I think it is a good thing it can be discovered in this way. I do not see why it should have been kept a secret. As a matter of fact I have repeatedly told Mr. Braund that it would be a good thing

if he threw the whole thing open, and I am very sorry that he has not done so.

MR. TREATT: Having heard the constituent parts of this alleged treatment, what opinion would Dr. Brose have of it now? What does he think of alum injected in large quantities? What is his opinion of that?

DR. BROSE: I do not know the exact effect of the alum alone, or of alum plus unknown substances, and if it only produces necrosis of the tissues which the alum reaches in a certain strength, which I presume would be the case—I imagine it would become weaker. I do not know. I only know he has removed big tumours and must have introduced bigger amounts of material, but I am as ignorant as any other member of the Committee.

CHAIRMAN: I do not think there is any ignorance on that matter. I think all the medical members will agree if I say, quite definitely—I am open to correction by any other member—that when you inject alum into the tissues you do not get it absorbed, and you induce automatically a slough.

DR. BROSE: Yes, after five minutes.

CHAIRMAN: Not a slough after five minutes. You will get coagulation of the tissues within ten minutes or quarter of an hour according to the way it can filter out into the tissues, and you will get a slough at the site of injection and that slough will have no relation whatsoever to the cancer. Will every medical member agree with that statement?

COMMITTEE MEMBERS: Yes.

CHAIRMAN: That is strictly in accordance with scientific fact! There is a motion before the Committee that my statement, including Dr. Taylor's certificate, together with any comments or explanations by Dr. Brose, be incorporated in the report. I ask Dr. Brose if there is anything further he wants to say in explanation.

DR. BROSE: I would rather like to hear what the statement is. I think it is important what statements are to be recorded.

CHAIRMAN: We are not going to record the lengthy letter to Sir James Elliott, or the one from Mr. Fraser, which are not material. The important point you have to satisfy the Committee on is, first, that you had no connection with Mr. Braund, that you know nothing about what it is, and you still maintain he has something up his sleeve.

DR. BROSE: I say there is a possibility of that. I do not maintain there is. It seems to me there may be something else, but I cannot make a categorical statement about its being one thing only. I smelt ammonia.

CHAIRMAN: Would you give us now—before we pass this motion, which will indicate what is to go into the report that I intend to finalize immediately—what you want to go into it as an explanation of the statement I have made and which implicates you very seriously?

DR. BROSE: The statement that he is using alum?

CHAIRMAN: The statement that he is using alum, and that you probably knew it.

DR. BROSE: You cannot say that. I did not know it. It is the first time I have heard alum mentioned in connection with cancer at all.

CHAIRMAN: We will have to leave that to the Committee to judge.

DR. BROSE: It is not a matter of judging; it is for you to prove it if you think that. It is not for me to prove my innocence before you gentlemen. I say, categorically, that I have not known, and do not know, what the substance is that is injected, and learned for the first time today that it may be alum or that alum may be a constituent of it.

CHAIRMAN: Do you still think it would be beneficial in the treatment of cancer.

DR. BROSE: I could not say that.

CHAIRMAN: You do not know?

DR. BROSE: I dare say that would be a field of research for somebody in animal experiments to try out the effect of aluminium on growths. I do not know.

CHAIRMAN: Would that be sufficient then as your reply?

DR. BROSE: Yes, I think that contains the essence of the thing.

(At this stage the Chairman put the motion. It was carried on the voices.)

CHAIRMAN: Do you wish to dissent, Dr. Brose?

DR. BROSE: Yes, rather.

Conclusions.

The following conclusions were adopted unanimously:

(1) The lack of cooperation by Mr. Braund and his actions which thwarted the examination of patients by the Committee are incompatible with his alleged desire to have his claim to cure cancer thoroughly investigated.

This attitude can be readily understood in the light of the information submitted by the Chairman at the final meeting of the Committee.

(2) Mr. Braund's alleged specific cure for cancer is the injection of alum into the subcutaneous tissues of patients, thereby causing necrosis of the tissues, with consequent separation of a slough which is claimed to be the actual cancer.

(3) From the examination of patients, claimed to have been cured of cancer by Mr. Braund, the Committee were unanimously of the opinion that in no case had such claim been substantiated.

Later knowledge regarding the use by Mr. Braund of injections of alum vindicates the Committee's finding, since such injections of alum can in no way affect the course of cancer.

(4) The exploitation of Mr. Braund's alleged cure amounts to an outstanding public mischief with international ramifications. It has lulled sufferers into a false sense of security, thereby preventing them from obtaining proper treatment at a curable stage of the disease, or, at least, affording them an opportunity to prolong their lives.

Acknowledgement.

It is desired to place on record the Committee's thanks and appreciation to Mr. Gray (Premier's Department) for his efficient services as Secretary of the Committee and to the typists of the Public Health Department who have willingly undertaken a considerable volume of additional typing to meet the requirements of this investigation. Special thanks are due to the Sydney Hospital for the facilities afforded the Committee to examine patients and especially to Dr. Sylvia Bray, of that hospital, for her cooperation in arranging for X-ray examinations and for her assistance in many other ways.

Very special thanks are due to Dr. H. B. Taylor, Government Analyst, for his excellent analytical work, which was invaluable and which enabled the Committee to make its findings definitely conclusive.

For and on behalf of all members of the Committee,

E. SYDNEY MORRIS, M.D., Ch.M., D.P.H., F.R.A.C.P., Chairman.
B. T. EDYE, M.B., Ch.M., F.R.C.S. (England).
A. J. COLLINS, M.B., M.S., F.R.A.C.P.
A. H. TEBBUTT, M.B., F.R.A.C.P.
A. T. NISBET, M.B., Ch.M., D.P.H.
M. BARRY, M.B., B.S.
G. H. B. DECK, M.B.
H. V. GILLIES, M.B.
V. H. TREATT, M.M., K.C., M.A., B.C.L., M.L.A.
C. C. LAZZARINI, M.L.A.
E. J. HALLSTROM.
H. L. BROSE, M.A., Ph.D., D.Sc.

G. M. GRAY, Secretary.

3rd April, 1948.

Appendix 1.

Report of the Medical Committee of Investigation Appointed by the Home Secretary to Investigate the Claims of Mr. G. A. Roberts that he had Discovered a Cure for Cancer.

The Honourable E. M. Hanlon, M.L.A.,
Home Secretary, Brisbane.

Sir: As you will recall, Mr. G. A. Roberts, in November, 1931, forwarded from Ayr (N.Q.) a telegram to the then Premier (the Honourable A. E. Moore) which reads as follows:

"Have discovered cure for internal and external cancer. Have completed tests to date after four years' study. All experiments have been carried out with strictest secrecy. Am prepared to give to Queensland Government free. Am late student of Prince Alfred College, Adelaide. Youngest son of Colonel Roberts. Please wire me advice regarding future action."

This telegram was forwarded by the Premier to the Cancer Trust, which in turn advised Mr. Roberts that it would gladly investigate the cure, providing the following conditions were complied with:

"The Cancer Trust is willing to investigate proposed cancer cures, but under no circumstances will such investigation be undertaken unless the inventor is prepared to disclose full particulars of his treatment. The Trust is prepared to regard such information as strictly confidential and to allow the inventor to take any reasonable precautions to safeguard his priority rights. No further investigation shall be made where the remedy is already known to medical science and known to be useless and/or dangerous."

Mr. Roberts then wired the Trust asking if he might give a demonstration in Brisbane, and the Trust replied that an investigation would be made if the above conditions were complied with. Mr. Roberts then published his claim in *Truth* newspaper. In December, 1931, he arrived in Brisbane, and

through the daily Press asked the Trust to define its attitude. It replied in the same terms as before, adding that if Mr. Roberts would disclose his formula, it was prepared to give any guarantee regarding secrecy. Interviewed by Mr. Roberts, a responsible officer of the Trust provided the same reply, which was not acceptable to him. From an address in Fortitude Valley he then began to use the daily Press to advance his claims. In June, 1932, Mr. Roberts registered a company called "Roberts Clinics Proprietary Limited", and many prominent citizens became shareholders. In the prospectus of this company appeared a challenge to the medical profession to investigate the cure in the following terms:

"I (G. A. Roberts) invite any doctor or group of doctors in Brisbane, or elsewhere, to furnish me with a medically diagnosed cancer case of their own choosing and I will treat the patient in their presence and then, immediately after the simple process of administering the injection, hand the patient back to their care. That done, *I guarantee that in seven to thirty-one days the cancer will have completely disappeared.*"

About this time Mr. Roberts, who was still advertising extensively in the metropolitan Press, requested the Government to undertake a test of his claims. You, as Home Secretary, referred the request to the Cancer Trust, which asked the Chairman of its Treatment Committee to draw up certain requirements, and to generally supervise the test. Mr. Roberts then declined to deal further with the Trust, which in turn referred the matter back to you as Home Secretary. Having agreed to conduct the test, the Government appointed the medical men originally nominated by the Trust, together with three medical men nominated by Mr. Roberts, and with the Public Health Commissioner (Dr. John Coffey) as government representative. One member of the Committee (Dr. J. V. Duhig) refused to act and gave adequate reasons for so doing to the Home Secretary. Mr. R. J. Fanning, of your department, was selected as Secretary to this Committee of eleven medical men.

The conditions laid down by you for the test were practically identical with those laid down originally for the Cancer Trust by the Chairman of the Treatment Committee with the exception that Mr. Roberts flatly refused to allow his preparation to be analysed by the Government Analyst even with the strictest safeguards regarding secrecy. (This compares with Mr. Roberts' claims in the prospectus of his company that his solution defied analysis by anyone!) The Trust, and subsequently yourself, as Home Secretary, decided to waive the condition of analysis in order to allow the test to proceed. By public advertisement in the Press, volunteers were called for from amongst those willing to undergo the treatment, and the first examination of sixteen patients took place at the Diamantina Hospital, and subsequently a further batch of volunteers was examined at Wattlebrae Hospital. Between 24th January and 26th February, thirty-seven patients were examined (twenty-eight males and nine females). Some of these patients were rejected by the Committee as being too advanced for treatment, or as suffering from some dangerous complication, or as not being cases of malignant disease at all. Mr. Roberts was given the right of final rejection in every case, and this right he exercised to such an extent that, of the twenty-one patients left after rejection by the Committee, he felt disposed to undertake the treatment of only three cases (this compares with Mr. Roberts' claim that he had discovered an infallible cure for cancer!). Moreover, the heavy percentage of rejections by him applied to nearly all cases of internal cancer and it was evident to the Committee from the outset of the test that he was studiously endeavouring to avoid the internal cases. Although the Committee had stipulated that the test should be applied to six cases (three cases of external and three cases of internal cancer), it was finally decided, owing to a shortage of suitable applicants, to agree to Mr. Roberts treating three cases (one external and two internal cases). These cases were treated in Wattlebrae Hospital, which was especially staffed and equipped by your department for the purpose. The first treatment was given on 1st February, and the only patient remaining under treatment was transferred to a private hospital, on the closing of Wattlebrae on 12th April.

In terms of the memorandum of agreement entered into by Mr. Roberts with this Committee and yourself as Home Secretary, we now beg to report that we have carefully supervised the test and that our findings are as follows:

CASE 1.—J.S., male, aged 47 years. Patient submitted by Mr. Roberts himself and found to be suffering from a large infiltrating tumour of the left cheek, but otherwise in good health. A small piece of the tumour was removed before the beginning of the treatment, and the pathologist reported it as an epithelioma, though diagnosed incorrectly by Mr. Roberts as a rodent ulcer. On 1st February treatment was begun by Mr. Roberts by the injection around the edge of the tumour of fifteen minims of the preparation employed by him. Some of the acid accidently flowed onto the patient's chin and neck and resulted quickly in a burn of the second degree. (This compared with Mr. Roberts' claim that his preparation is harmless to healthy tissues!) About seven days later, as the Committee expected, the escharotic fluid began to eat out the main mass of the tumour which then commenced to necrose and come away in sloughs. From day to day the resulting ulcer was treated by painting upon it the escharotic fluid, and by the application of moist dressings. This continued until 7th March, when a further microscopic examination of a strip of the edge and floor of the ulcer showed that while the edge was still definitely malignant the tissue in the floor of the ulcer had, as a result of the constant stimulation by the escharotic employed, *been rendered more intensely malignant than was the original tumour.* In addition to this sinister development the patient, who before the beginning of treatment was able to easily open his mouth, was (and still is) unable to separate his teeth by more than half an inch. The floor of the malignant ulcer is now firmly infiltrating the muscles of mastication—the facial nerve on the affected side, which previously was quite intact, has been destroyed, leading to paralysis of one side of the face, and the lower jaw at its angle is already involved by the cancerous process. This involvement of the bone by the cancer gravely influences the ultimate outlook for this patient's life. On these grounds we are unanimously of opinion that this patient is now definitely worse than at the beginning of the course of treatment, in spite of the fact that the main mass of the tumour has been sloughed out by the caustic employed, a result which could have been achieved by surgical diathermy in a fraction of the time.

CASE 2.—Mrs. S.M., aged 70 years. This patient had cancer of the lower end of the rectum. Mr. Roberts, having agreed that she was a suitable case for his method of treatment, began his ministrations on 17th February by injecting 15 minims of acid through a needle into the lower aspect of the left buttock via the perineum. An abscess soon formed and, after causing much pain and burning sensation locally, finally burst into the rectum above the growth, as well as on the surface through the perineal needle puncture. In addition to the pain the patient's suffering was intensified by the formation of this fæcal fistula. Examination of the growth now caused extreme pain, and showed that the cancer had spread right around the bowel wall. After four weeks this patient was no longer visited by Mr. Roberts, who abandoned the case after his admission to the Medical Committee and yourself on 6th April that he could do no further good by continuing treatment. The patient was finally discharged to her own home on 12th April, and her early demise is to be expected. In regard to this case we are unanimously of opinion that the treatment employed by Mr. Roberts has done this patient no good whatever—a fact which Mr. Roberts has openly admitted—but that, on the contrary, it has stimulated the cancer to make more rapid headway than if no treatment whatever had been employed.

CASE 3.—Mr. W.L., aged 57 years. This patient had had his case diagnosed as cancer of the body of the stomach in Casino, Lismore and Ashfield (New South Wales) as well as in Brisbane. He was considered by Mr. Roberts as a suitable case for treatment. Accordingly on 2nd February a fine stomach tube was passed down the gullet, and through this Mr. Roberts slowly injected a small quantity (about 15 minims) of highly diluted acid. The tube was then clipped and slowly withdrawn. Mr. Roberts then ordered that no further treatment should be employed in this case for one month, at the end of which time he would require a further X ray of the stomach. An experiment conducted by me with a precisely similar tube proved that the quantity of solution employed by Mr. Roberts just sufficed to fill the small stomach tube. This on being clipped and withdrawn slowly still contained all the solution injected by Mr. Roberts, so that in reality little of it actually entered the stomach cavity of the patient. Moreover, this Committee quite fails to see how any solution injected haphazardly could possibly cause the healing of an extensive cancerous invasion of the stomach walls, as shown by the X-ray films in this case. At the end of six weeks a second "injection" was given by Mr. Roberts to the patient. The tube was again clipped before removal, and its contents subsequently collected from the tube itself, by the Committee, and submitted to the Government Analyst, whose report showed that it was a harmless neutral preparation and quite unlike the caustic acid solution which Mr. Roberts claimed to use in his treatment of cancer. Between the two injections a small painless lump developed in the abdominal wall of this patient. It was removed under a local anæsthetic by Dr. Casey, of the Brisbane General Hospital staff, and submitted to the pathologist. His report showed that the mass was a small secondary cancerous nodule showing stomach glands. This sinister development proved, of course, that the disease had left the stomach to infect distant parts of the body. At the end of a month the stomach was again X rayed as requested by Mr. Roberts, and the films showed that the disease had advanced so rapidly that practically no healthy stomach remained. Meanwhile the patient had lost weight, strength and colour and was so obviously going dowhill that at the Committee meeting at Wattlebrae on 6th April Mr. Roberts, on being questioned, openly admitted that he could do nothing further with this patient, who on 13th April was discharged to his own home in New South Wales.

With regard to this case we are unanimously of opinion that the treatment employed by Mr. Roberts did not confer the slightest benefit upon this man. On the contrary, we are not certain that the first injection on 2nd February did not stimulate the neoplasm to grow more rapidly. We feel that the harmless injection on 17th March did not influence the progress of the growth in any way.

At the conclusion of this report we would like respectfully to bring under your notice certain observations we have made during the conduct of this test.

(*a*) *The Qualifications of Mr. Roberts to Treat Cancer.*—When this gentleman first came to Brisbane he used visiting cards on which he was described as "Dr." Roberts. Again, in the prospectus of his company, he deliberately states that he spent four and a half years as a medical student at Adelaide University. We are of opinion that Mr. Roberts has never at any time been a medical student at any university and that he is profoundly ignorant of all aspects of the cancer problem, in which he describes himself as a specialist. During the conduct of the test we had many opportunities of observing Mr. Roberts' ignorance of medicine and chemistry. For instance, he did not know the meaning of many simple medical terms, and confused cubic centimetre (15 minims) with a

single minim. Moreover, we entirely disbelieve Mr. Roberts' statement that he spent four years in cancer research in Ayr (N.Q.).

(*b*) *The Fairness of the Test.*—During the conduct of the test no attempt whatever was made by the nursing staff or any member of the Medical Committee to in any way interfere with Mr. Roberts' method of treatment, and we are able to assure you that so far as this Committee is concerned the test has been conducted with absolute fairness. No attempt has been made at any time to foist incurable or hopeless cases upon Mr. Roberts, nor was any attempt made to deprive volunteers of their right to be treated by him if they so desired. We class as utter nonsense the statements made in certain metropolitan newspapers by, or on behalf of, Mr. Roberts that any member of this Committee had endeavoured to "abduct" him while he was away recently in North Queensland. We think it will be conceded that in the face of many innuendoes and false charges my Committee have borne themselves with extreme patience.

(*c*) *The Propriety of Unqualified Persons being Allowed to Treat Cancer.*—From a public health aspect we view with apprehension the facility with which a layman has been able to float a company to commercialize the treatment of a fell and sinister disease like cancer—a project in which Mr. Roberts has been able to financially interest certain well-known citizens of the State as well as enlist the moral support of at least one metropolitan newspaper.

We are unanimously of opinion that the method of treating even external cancer by acids or other escharotics as exploited by Mr. Roberts is based on wrong premises, and is a sham and a fraud, and that any attempt to treat internal cases by the injection of any such substance is nothing short of a criminal act. Moreover, it is precisely the form of treatment long since condemned by cancer research workers the whole world over. The following extract (from "Cancer, What Everyone Should Know About It", by Dr. J. A. Tobey), published by the American Society for the Control of Cancer and reprinted by the Queensland Cancer Trust, sums up very aptly in our opinion the fraudulent nature of this type of treatment:

"With the decline of patent medicines there has arisen a new racket in the cancer cure field. The swindler who preys on patients (in America) has now turned to the management of a hospital or clinic as a cloak for his activities. Victims are lured to these institutions by means of prospectuses which proclaim that the owner possesses a remarkable remedy, invariably a secret and mysterious one, for cancer. The circulars, often elaborately printed, contain startling pictures of skin blemishes, alleged to be malignant.

"The cure usually consists of repeated applications of an escharotic, which means a caustic or corrosive substance with a zinc or lead base. A lesion that may only be a benign blemish is eaten out by the caustic, the patient pays his hard-earned money, and comes away convinced that he has escaped a horrible death. Actually he may have wasted valuable time and cash on a worthless remedy for a harmless condition. If, on the other hand, he really had a cancer, he has been swindled also, since chemicals cannot cure cancer. Lulled into a false sense of security, he discovers finally that he is worse off than before.

"Nothing is easier to obtain than a testimonial. During the first flush of enthusiasm after a new 'cure' a gullible patient subtly urged on by a high-pressure salesman is not only willing but often eager to testify to its efficacy. A few years later, or possibly much sooner, he dies of the disease. The cemeteries are full of such testimonial givers."

In conclusion, Sir, we wish to express our appreciation of the valuable assistance rendered throughout the test by Dr. Casey, of the Brisbane Hospital staff, and of the skill and care exercised in the treatment of the patients at Wattlebrae by Matron Taylor and her staff; to Matron de Mattos, of Toxteth Private Hospital; to Mr. R. J. Fanning (Secretary of the Committee), who has performed a difficult and delicate task with great credit; to Mr. Waller (shorthand reporter); and lastly to yourself, as Home Secretary, for the fair and absolutely impartial attitude you have taken up during the test, and for the equipment and facilities you have provided for its conduct.

For and on behalf of the following members of the Committee of Investigation, viz., Carl Brunnich, M.B., Ch.M., Robt. Grey, M.B., Ch.M., Wm. P. Kelly, F.R.C.S.I., D.P.H., G. C. Taylor, M.B., Ch.M., H. S. McLelland, M.B., Ch.M., F.R.A.C.S., John Coffey, M.B., F.R.C.S.E., D.P.H., V. McDowall, M.B., Ch.M., B. L. W. Clarke, M.B., B.S., D.M.R.E., Neville Sutton, M.B., Ch.M., F.R.C.S.E., F.R.A.C.S., and J. C. Hemsley, M.B., B.S., F.R.A.C.S.,

I am, Sir,

Obediently Yours,

(Sgd.) L. M. McKILLOP, M.B., Ch.M., F.R.C.S.E., F.R.A.C.S., F.A.C.S., Chairman.

Brisbane,
28th April, 1933.

Signatures of Members of Committee: C. Brunnich, Robert Gray, W. P. Kelly, G. C. Taylor, H. S. McLelland, John Coffey, V. McDowell, B. L. W. Clarke, Neville E. Sutton, J. Cameron Hemsley.

Appendix 2.

(Extract from *New Zealand Medical Journal*, Volume XXXVIII, No. 204, April, 1939, page 120.)

The Koch and Baker Cancer Cure.

In August of last year the Minister of Health, the Hon. Peter Fraser, appointed a Medical Committee to investigate a special treatment for cancer which was to be used on patients of Dr. Ulric Williams at Wanganui. The Committee was set up at the request of Dr. Ulric Williams, who had associated with him Professor Henry Brose, of Sydney, and Mr. McCulloch, who describes himself as a naturopath.

This Committee, consisting of Sir James Elliott, Chairman, Dr. H. Hardwick Smith, Dr. T. R. Ritchie and Dr. J. O. Mercer, was unable, for reasons which appear in the report, to complete its investigations on the cases selected by Dr. Williams and his colleagues. Nevertheless, sufficient data was obtained to enable the following report to be presented to the Minister:

Report of Government Investigation Committee.

The members of the Committee of Investigation approached this inquiry with an open mind and the desire that some efficacy might be disclosed in these cures, even although one part of the treatment, namely, by Koch's fluid, had been investigated at various times in America during the past twenty years.[1]

For the purpose of the investigation the Committee proceeded to Wanganui on the 31st August, 1938, and carefully examined fifteen patients treated by Mr. McCulloch under the supervision of Dr. Ulric Williams. Professor Brose was, so he informed us, chiefly interested in this method of treatment as he was anxious to test in these cases a method which he had devised for testing the blood of patients to find if they had cancer or not, and if so, the progress of this disease. It is right to say that these tests used by Professor Brose are not generally accepted.

Of the fifteen cases, the Committee decided that twelve were clinically cases of cancer, but in six of these histological proof of the diagnosis was lacking. Subsequently two of these latter were proved to be cancer by pathological examination. The remaining three cases were not, in the opinion of the Committee, suffering from malignant disease.

All the patients that we saw on this occasion had already been treated, so that we were given no opportunity of actually seeing the treatment given or of examining the fluids which had been used for the treatment.

We were especially interested in four cases of cancer of the breast which had been treated by local injections of fluid round the periphery of the tumour ten days prior to our visit, and we were informed that they had also been given an intravenous injection on the 30th August.

The results as we saw them on the 31st August were as follows:

> In each case the whole of the breast and a varying but extensive surrounding area was completely gangrenous, black and malodorous. All these patients had undergone the diet treatment previously.
>
> No temperature charts or case reports were available, but the Investigation Committee was able to get in some instances reports and records of cases from hospitals and medical practitioners in other parts of New Zealand where these patients had received advice or treatment. This was done with the knowledge and consent of Dr. Williams.

The Committee of Investigation was invited to see these four cases of breast cancer again on the 14th September, because, we were informed, Professor Brose and Mr. McCulloch were leaving for Australia and were desirous that we should see the improvement that had taken place in the condition of these patients. We were told at this time that the treatment given was the injection of Baker's fluid locally in the breast cases, and, in addition, intravenous injection of Koch's fluid in all cases. Mr. McCulloch gave the Committee the impression that Baker's fluid (in the breast cases) had a selective and exclusive action in destroying cancerous tissue only and that Koch's fluid searched out all cancer cells throughout the body wherever they might be and destroyed them.

In the opinion of the members of the Committee the general condition of the four patients showed no improvement. Indeed, two of the four cases were markedly worse. In all cases the gangrenous breasts had sloughed away together with the surrounding tissue, extending in one instance from the centre of the breast bone to the posterior margin of the arm-pit and exposing a large raw area of breast muscle. In one case healing had already commenced to take place from the margins of the raw area. The other patients showed no signs of healing at that early stage and would require a considerable period for the healing of the large areas. There was a great amount of mutilation.

In three of the four breast cases the diagnosis of cancer was confirmed microscopically by an examination of the separated breast tissue.

It proved that this second visit of investigation on the 14th September was also to be our last. Quite unexpectedly Dr. Ulric Williams sent word that our investigations were to cease. You will remember, sir, your efforts to induce Dr. Ulric Williams to reconsider this decision. He declined to do so. His last communication on the subject, under date 16th December, states:

"Since both Baker and Koch have written to me in the strongest terms declining to submit their products to test in their absence, I have no alternative to breaking up negotiations with your Committee."

The Committee, although thwarted in its investigation in this way, has seen reports of investigations made into both the Baker and Koch treatment in the United States of America

[1] The cures referred to are injections by the methods of Koch and Baker, which have already been widely advertised in the United States of America.

under more favourable conditions and these reports have been very condemnatory of the treatment.

As a result of our observations and study of the cases seen at Wanganui, we can make certain conclusions.

We have no doubt that Baker's fluid is a powerful sclerotic agent and can cause great local destruction. Compared with surgical treatment, destruction by a sclerosing or caustic fluid seems like a reversal to the treatment in the Middle Ages, causing unnecessary mental distress, delayed healing and increased risk of septic infection. We have had no evidence whatever to show that Baker's fluid has any selective powers in the destruction of cancer cells. There is no real difficulty in removing the breast surgically or by crude destruction, but this is very far from a cure of the breast cancer and may delude the public mind. The danger of death from cancer of the breast lies in the great tendency of the breast cancer, if not treated early and by substantiated methods, to spread to the arm-pit, inside the chest, into the bones or elsewhere.

From our general knowledge and from the observations we were permitted to make in investigating the cases at Wanganui, we have no reason whatever to suppose that Koch's fluid has any effect in curing generalized cancer.

All the evidence available, without exception, convinces your Committee that the claim cannot be substantiated that the treatment which we saw practised at Wanganui has any merit as a cure or palliative for cancer. The claim that a cure has been found for cancer should never be made and promulgated until prolonged tests have shown that the claim is a reasonable one; otherwise patients who might be cured or their lives greatly prolonged by surgical or radiological means, because of their dallying with vaunted cures such as Baker's and Koch's, have thereby allowed their disease to advance from the early stages, when it can be treated by recognized and proved methods. This is all the more important when such alleged cures as Baker's and Koch's are sold as commercial products and when cancer patients are called upon to pay in some cases high fees for the treatment.

Your Committee considers that it is in the public interest that a statement should be made that our hope of finding anything useful in the treatment by Dr. Williams and his colleagues has proved illusory.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Children's Hospital, Carlton, Melbourne, on November 19, 1947. The meeting took the form of a series of demonstrations by members of the honorary medical staff of the hospital and was combined with the monthly meeting of the Melbourne Pædiatric Society. Part of this report appeared in the issue of May 22, 1948.

Streptomycin in Treatment of Meningitis.

DR. STANLEY WILLIAMS showed a series of patients illustrating the use of streptomycin in the treatment of meningitis.

Tuberculous Meningitis.

The first patient was a girl, aged seven years, who, when admitted to the Children's Hospital under the care of Dr. J. W. Grieve, had been irrational and dehydrated, and had later developed papilloedema. The cerebro-spinal fluid contained 170 cells and 620 milligrammes of chloride per 100 millilitres. A culture from the cerebro-spinal fluid was prepared by Dr. Reginald Webster, who reported four weeks later that *Mycobacterium tuberculosis* had been isolated in the culture. Administration of streptomycin was commenced on July 13 in a dosage of 75 milligrammes given intrathecally each day and 100 milligrammes given intramuscularly at three-hourly intervals. Six days later Dr. Reginald Hooper performed a ventricular puncture and inserted 100 milligrammes of streptomycin into the left lateral ventricle. The procedure was repeated next day on the right side, and on the following three days on the left side. Improvement in the child's condition commenced nineteen days after she had first received streptomycin and had continued until at the time of the meeting she was walking about and appeared well. The intrathecal administration of streptomycin was continued for ninety-nine days, and stopped on October 20. Seven days later the cerebro-spinal fluid contained 14 lymphocytes and 720 milligrammes of chloride per 100 millilitres. The child was still receiving streptomycin by the intramuscular route.

Another girl, aged eight years, shown by Dr. Williams, had been admitted under the care of Dr. A. P. Derham. She was suffering from tuberculous meningitis and was similarly treated with streptomycin. *Mycobacterium tuberculosis* was isolated from the cerebro-spinal fluid collected on July 10, 1947, and treatment was commenced on July 11. After one hundred days' treatment, streptomycin was stopped for seven days and the cerebro-spinal fluid was found to contain 210 cells, of which 60% were polymorphonuclear leucocytes, and 700 milligrammes of chloride per 100 millilitres. As the child's general condition was not completely normal, streptomycin was again administered intrathecally and intramuscularly and was still being given one hundred and twenty days after the commencement of treatment. The child was well, though thin and easily tired.

Dr. Williams commented that it was too early to state that either child was cured, but the first patient had shown truly miraculous improvement, whilst the second child also gave promise of a satisfactory result. There were no deafness or other features to suggest toxic effects from the streptomycin.

Meningitis due to Hæmophilus Influenzæ.

Dr. Williams then showed two children who had suffered from influenzal meningitis as examples of patients gaining satisfactory results from the combined treatment of antibacterial rabbit serum, sulphadiazine and streptomycin. It was emphasized that to obtain the best results early diagnosis was essential, and the most important single form of therapy was the antibacterial rabbit serum. Streptomycin had been used in a dosage of 50 to 75 milligrammes given intrathecally and 75 to 100 milligrammes given intramuscularly at three-hourly intervals and continued for ten to thirty days. Dr. Williams pointed out that the shorter time the streptomycin was given the better, provided that it was compatible with a satisfactory response; that response was obtainable in most cases in ten to fifteen days. There had been no indications of severe toxic reactions due to streptomycin, but those were sometimes difficult to assess, because the meningitis itself could produce sequelæ indistinguishable from a toxic cerebral reaction. In the patients shown there were no toxic sequelæ of the meningitis or of the therapy.

Development of the Breast.

DR. H. BOYD GRAHAM gave a lecturette, illustrated by means of 35 millimetre film strips and slides, which dealt with the embryology of the breast and the mechanism of certain developmental anomalies. The cases of a small girl with temporary precocious mammary over-development and of an infant boy with absence of all traces of breast tissue on the left side were the subjects of special reference.

Skeletal Deformities in Childhood.

DR. L. P. WAIT, DR. GWEN DONALD and DR. PHILIP BROWN presented a series of patients illustrating skeletal deformities in childhood.

Morquio's Disease.

The first patient was a girl, aged six years, suffering from Morquio's disease. The child's mother suffered from the same condition, her height being three feet four and a half inches, but there was no family history of dwarfism, the grandparents were of normal height and the mother's three brothers were of average height. The child's father had a height of five feet three inches. The child was born by Cæsarean section at the Women's Hospital, Melbourne; she appeared to be of normal size and grew normally. Her mother had first recognized similarity to her own condition when the child commenced walking. The patient was now an exact replica of her mother. The facies and head of both were normal, which was not the case in achondroplasia. The skeletal changes of both as shown by X-ray examination were similar and were characteristic of the osteo-chondral dystrophy of Morquio. X-ray examination of the child showed that the skull was normal. In the spine there was flattening of all the bodies of the vertebræ (platyspondylia). Examination of the long bones revealed shortening of all "tubular" bones with thick cortex, and "mushrooming" of the lower ends of the femora and of both ends of the tibiæ; the proximal ends of the humeri were underdeveloped; the glenoid cavity was small; the articular ends of the metacarpals and metatarsals were rarefied, contrasting with the thick cortex of the shafts of those bones; the carpal and tarsal bones showed uniform rarefaction. X-ray examination of the mother showed similar changes, excepting in the spine, the bodies of which were normal in appearance, though there was gross scoliosis; both hip joints showed gross osteoarthritis. By way of treatment, thyreoid extract had been tried without effect. A course of "Perandren" was given parenterally without any obvious change. It was-

thought that the child, by her present rate of growth, might be taller than her mother. Both mother and child exhibited more than average intelligence.

Osteogenesis Imperfecta.

The next patient, an Italian girl, aged fourteen months, had been one of twins; the other died soon after birth. Deformities of the limbs were noticed by the parents. When the child first attended hospital she had gross deformities of the limbs, bilateral wrist drop (fractures of the humeri were noted in X-ray films), soft skull bones which were easily indented with the fingers, and very blue sclerotics. The last had become less obvious. X-ray examination of the skeleton showed the characteristic signs of *osteogenesis imperfecta* with multiple fractures; the bones of the skull vault were extremely thin; the long bones had a rarefied appearance. It was stated that over the past few months her condition had improved. The skull had become quite hard, but she had multiple deformities particularly of the lower extremities. There was still evidence of bilateral wrist drop.

Gargoylism.

The next patient, a girl, aged two and a half years, although not a typical gargoyle, had many of the symptoms and some of the radiological findings of that disease. She had first attended hospital when aged six months. The mother had been worried by her delay in development as compared with her other child, who was aged six years and was quite normal. She suckled with difficulty, was late in taking notice and in trying to sit up. There was no family history of dwarfism. Examination revealed a child with a depressed bridge of the nose, coarse broad facies, a dry, harsh skin and a corneal opacity of the right eye which was probably traumatic in origin. Her cry was harsh. There was some rigidity of the knee joints. Although the abdomen was protuberant there was no hepato-splenomegaly. Skeletal survey by X rays showed that the *sella turcica* was elongated and shallow; the skull bones had a ground-glass appearance; there was no "beaking" of any vertebræ in the lower dorsal and lumbar regions. When the child was aged fifteen months, examination of the hip joints showed delayed development of both femoral heads. Later, when she was aged two years, X-ray examination of the hip joints showed bilateral *coxa vara* with some irregular mineralization of the femoral heads. In the humeri there was slight spindle-shaped swelling of the upper portion of the shafts. The child's progress had been slow, but there had been improvement. The blood cholesterol level was 100 milligrammes *per centum* (normal 100 to 200 milligrammes *per centum*). She had been having thyreoid one grain twice a day and the mother believed that that had helped.

Amyotonia Congenita.

The last patient in the series was a boy, aged three years, who had first attended the hospital when aged ten months. He had been a normal full-term baby with a birth weight of nine and a half pounds. At the age of ten months he weighed twenty pounds. He was the second child; the other was normal. The pregnancy had been normal. The child was never breast fed. He was as a rule generally uninterested, but smiled occasionally. He could not sit up or hold his head up. Two months later, at the age of one year, he had shown some improvement, but could not sit up, nor would he play with toys. He was a dull, expressionless, grossly atonic child. His head circumference was eighteen inches. X-ray examination of the skull revealed no abnormality. When the child was aged about two and a half years there was still gross hypotonia, but he had shown definite improvement and looked and behaved more intelligently. In the past year progress had been more rapid and the prognosis was reasonably good. He was having massage and educational exercises. The child had earlier been regarded as mentally deficient by other medical men. The comment was made that the most important but perhaps the most difficult part of the management was the advice to parents, as without encouragement by the medical officer good results could not be attained.

Nutritional Anæmia in Infancy.

DR. JOHN COLEBATCH presented a group of four patients illustrating the ætiology and treatment of various types of nutritional anæmia in infancy. He emphasized that the anæmia in each case was due to deficiency of such hæmopoietic factors as iron, folic acid and protein. The deficiency might be purely dietetic in origin, but more often it was caused or conditioned by maternal malnutrition or prematurity with a low fœtal store of hæmatopoietic factors, or by the increased demands of rapid growth, of hæmorrhage, of repeated infections *et cetera*. Whatever the mechanism of its production, the anæmia was essentially a deficiency disease and as such it had to be regarded as preventible. It was considered that this fact deserved wider recognition, especially by those undertaking the care of pregnant women and premature babies. It was also a practical point that in none of these cases had anæmia or pallor been mentioned by the mother as a symptom.

The first patient, a twin boy, born three weeks prematurely, had been a difficult feeder from birth, and was five pounds underweight. He had a severe functional dysphagia for solid and semi-solid foods and consequently his diet had been almost devoid of meat, green vegetables, fruit and bread. He had never been given medicinal iron. When seen at the age of twenty-one months his red blood cells numbered 4,240,000 per cubic millimetre and his hæmoglobin value was 45% (6·5 grammes per 100 millilitres), the colour index being 0·53. Other investigations including X-ray examinations with a barium bolus revealed no abnormality. Treatment included iron (ferrous sulphate, four and a half grains daily), antispasmodics, and a balanced diet. The dysphagia had been only partially relieved, yet the blood picture had returned to normal and the weight had increased at a normal rate. The anæmia in this case had been due to deficiency of iron in the diet, the dysphagia having caused refusal or vomiting of most of the iron-containing foods. It was significant that the twin brother, with identical birth weight, had never been ill or anæmic.

The second patient, a female baby, aged six months, had never gained weight satisfactorily and was subject to cough and frequent "colds". Her mother had had toxæmia of pregnancy for which she had spent ten weeks in hospital, deprived of meat and eggs, and without medicinal iron. At the end of the seventh month of the pregnancy the child had been delivered by Cæsarean section, and weighed two pounds fifteen ounces. At the age of ten weeks she had been discharged from hospital, since when she had been fed on weak mixtures of cow's milk with added vitamins but no iron. That history led one to suspect a gross deficiency of both iron and protein. Examination revealed that she was two pounds underweight, with pallor, coryza and koilonychia. The red blood cells numbered 3,000,000 per cubic millimetre, the hæmoglobin value was 36% (5·2 grammes per 100 millilitres) and the colour index 0·6. Treatment included iron (ferrous sulphate, up to four and a half grains daily) and an increased protein intake provided by stronger milk mixtures and by mixed feeding. As a result her blood picture had reached normal limits and she had gained nine pounds in her second six months of life.

The next patient, a male baby, aged five months, was brought to hospital for treatment of a nævus on the lip. He had been born "at six months", when he weighed three pounds. He had been given expressed breast milk for the first two and a half months and then had been breast fed, with no additional vitamins, iron or protein. He had gained weight rapidly—eight pounds in five months. Examination revealed, *inter alia*, beading of the ribs and Harrison's sulci. The red blood cells numbered 4,370,000 per cubic millimetre, the hæmoglobin value was 60% (8·7 grammes per 100 millilitres) and the colour index 0·69. There was radiological evidence of rickets at the wrists, knees and ankles. Treatment with iron, educational diet and vitamins led to a rise of hæmoglobin value to 100% (14·5 grammes per 100 millilitres) and to disappearance of the rhachitic signs. The anæmia was due to iron deficiency caused by the low fœtal store of iron associated with prematurity and by the increased demands made by the baby's rapid growth. Dr. Colebatch commented that the patient gave support to the view that for premature babies breast milk alone could not provide an adequate supply of nutritional factors such as iron, vitamins and protein.

Dr. Colebatch's fourth patient was a girl, aged two years, who had been diagnosed in her first year as suffering from fibrocystic disease of the pancreas with steatorrhœa and stunting. Although there had been considerable progress under treatment, the blood had shown a persistent mild degree of anæmia of slightly macrocytic type associated with gross neutropenia. A typical blood picture revealed 3,000,000 red blood cells per cubic millimetre, a hæmoglobin value of 63% (9·1 grammes per 100 millilitres), a colour index of 1·05 and 5000 white blood cells per cubic millimetre, of which neutrophile cells formed 11% (550 per cubic millimetre). Folic acid in large doses (60 milligrammes *per diem*) had produced a sharp rise in the number of neutrophile cells to above the normal range, and a less dramatic rise of red blood cells and hæmoglobin. The child appeared to suffer from defective absorption of folic acid, but further trials were being made to elucidate that point.

Sequelæ of Erythroblastosis Fœtalis.

Dr. Colebatch also presented a group of four patients showing the late results of treatment of *erythroblastosis fœtalis* in babies who had survived. The demonstration was illustrated with colour photographs and with sections prepared by Dr. Reginald Webster to show the pathological changes in the liver in the disease.

One baby, who had been jaundiced for five weeks, had had a persistent normochromic anæmia of minor degree, until at the age of four and a half months her protein intake had been increased by strengthening the milk feedings. After that change in treatment the weight had increased rapidly and the blood picture had become normal. Another baby, who had had severe *erythroblastosis fœtalis* with jaundice for six weeks, presented signs of nuclear damage (cerebral and perhaps cerebellar) and defective teeth. The third patient, a girl, aged three and a quarter years, had been jaundiced for four and a half months after birth with gross hepato-splenomegaly. She had progressed well on a diet of high protein content with gradual reduction in the size of her liver and spleen. The last patient, a girl, aged two years, had had severe *erythroblastosis fœtalis* with hepato-splenomegaly but without persistent anæmia. When aged eleven months she had been readmitted to hospital with vomiting, anorexia, stunted growth, hepatomegaly and macrocytic hæmolytic anæmia. Frequent blood transfusions had been required for the next six months. She had then been placed on a diet with a high protein content, on the assumption that liver damage was the most probable cause of the macrocytic hæmolytic anæmia. After that no further transfusions had been required and the rate of gain in weight had been doubled. Dr. Colebatch said that the child was under the care of Dr. H. Boyd Graham, who had invited Dr. Colebatch to present her to the meeting.

This group of cases will be reported in detail at a later date.

(To be continued.)

Correspondence.

STANDARDS AT THE UNIVERSITY OF SYDNEY MEDICAL SCHOOL.

SIR: In his letter published in THE MEDICAL JOURNAL OF AUSTRALIA, May 8, 1948, Mr. Howard Bullock quotes three extracts from one of the Sydney daily papers with very little comment. I wish to point out that the remarks attributed to me were not made in my capacity as President of the Medical Board of New South Wales, but as a private individual who has some knowledge of the standards in Sydney Medical School. The remarks published were not quite as I gave them nor in sequence, but such orientation seems to be a privilege of the lay Press. I have been intimately associated with the Anatomy and Surgery Departments for some thirty-seven years as well as with the Post-Graduate Committee since its inception, and it is only in the last year or two that I have withdrawn from examining.

There is no need to comment on either the physical fitness or intellectual capacity of the present-day medical students, for these standards have improved over the last thirty years, and it is just as well this is so to enable them to cope with the hurly-burly of present conditions at the university. Present-day students and graduates have not had the opportunity for that sound basic training in either their science or clinical years that was available even ten years ago. Professors, lecturers and demonstrators are all overworked today, and it is impossible for them to exercise that detailed and intimate contact between teacher and student that used to exist and which was of inestimable value. Some subjects have been curtailed, there is no practical operative surgery for lack of cadavers, and the position with training in midwifery is serious.

The point I wish to make is that because of overcrowding the medical student today cannot be given the same individual tuition as used to be the case, nor has he the same facilities for practical experience in either the science or clinical years, and so much is this the case that without outside coaching it is difficult for an average student to pass the examination in many subjects of the curriculum. At the end he may pass his examinations, but is by no means as adept as he should be in the practical application of his knowledge. There is a provision in the New South Wales *Medical Practitioners Act*, 1939, making one year's residency at a hospital obligatory before registration by the Medical Board, and the reason for such a provision should not be difficult to understand. However, every year this provision is suspended as there are not enough hospitals to take all recent graduates, and the pity of it is that such appointments go to the good men of the year, whilst those not so good who really need this extra training are turned out to acquire it as best they can. The position is being made worse in the clinical years by the steadily increasing number of European doctors applying for attendance at the Sydney Medical School for the last three years of the course, after which, if they pass the examination, they are candidates for registration in New South Wales.

As Sir Alan Newton remarks, the remedy lies with the New South Wales Government, as the Sydney Medical School is, I think, unique in that it cannot exercise any selection of students and must take all who apply provided they have matriculated at the requisite standard, irrespective of whether they can be taught efficiently or not.

Sir Hugh Cairns told us recently that at Oxford they select a strictly limited number for entry to the Medical School and that number is restricted still further for entry to the clinical years, so that adequate practical training can be assured.

That graduates realize their own shortcomings is made evident by the extraordinary demands made on the Post-Graduate Committee for tuition in many and varied subjects as well as in the General Revision course. If Mr. Bullock can advance some constructive ideas or take some active part in trying to better the training of students and young graduates his help and advice would be welcomed by all concerned with this difficult position.

Yours, etc.,

225, Macquarie Street, Sydney,
May 17, 1948.

HUGH R. G. POATE.

THE PHARMACEUTICAL BENEFITS ACT.

SIR: Judging by the article in *The Sydney Morning Herald* of today the Council is still opposed to the Government free medicine scheme.

I profoundly hope that it will not go further in its opposition to the bill than requesting members of the British Medical Association not to use the formulary, without threats or attempts at compulsion: for it is ultimately a matter of conscience. Members of Council have their consciences offended by the thought of using a formulary which they have not yet seen; the consciences of a number of loyal members of our Association would be offended by being compelled not to use it, even when it supplied the needs of their patients. They would feel that it was morally wrong to force a patient to spend, unnecessarily, money on medicines which was urgently needed for food; certainly no patient who is on or near the basic wage can pay for expensive drugs without depriving his family of some of the necessities of life.

To me the objections of the Council are not weighty enough to justify withholding from the poorer members of our community even this slight relief from the burden of mounting costs. All of us who have worked in hospitals have prescribed from a formulary; all who attend lodge patients prescribe from a much more limited one; yet I cannot remember that Council has ever objected to these.

In my lodge work I have frequently had to tell a patient that proper treatment called for a prescription outside the formulary, for which he would have to pay, and I have never met with any protest; inadequacies in the Government's formulary could easily be met in the same manner.

I hope that the Council will not attempt to use compulsion on those of us who, for the sake of our patients, feel bound to use the formulary whenever it meets their needs. As one's own considered judgement of what is right is more compelling than the orders of any Association, some of us would be forced to resign rather than conform.

Yours, etc.,

Katoomba,
May 6, 1948.

E. P. DARK.

SIR: It is strange that after many years of membership Dr. E. P. Dark is apparently unaware that the British Medical Association is not a trades union and has no power to compel its members to use or not to use the Commonwealth Pharmaceutical Formulary.

Dr. Dark's statement that "members of Council have their consciences offended by the thought of using a formulary which they have not yet seen; . . ." is untrue and misleading, as he would have found out if he had read carefully

the issues of THE MEDICAL JOURNAL OF AUSTRALIA of September 6, 1947, and May 1, 1948.

In the report of the Federal Council meeting of July 17 to 22, 1947, which appears in the journal of Sepember 6, 1947, the following statement appears, *viz.*: "After the conference [with the Minister for Health] the representatives of the Federal Council met to discuss what action should be taken in regard to the request of the Minister for advice on the following three points: (*a*) deficiencies of the Commonwealth Pharmaceutical Formulary . . . Dr. A. J. Collins and Dr. F. W. Carter were appointed a subcommittee to examine the Commonwealth Pharmaceutical Formulary with the right to consult and to submit a report to the committee. . . ." The report of the meeting of the Federal Council in the journal of May 1, 1948, includes a letter from the Minister for Health of August 13, 1947, from which I quote: "Following on the April conference I received your letter of 29th May last forwarding comments on the 1944/45 Commonwealth Pharmaceutical Formulary. . . ." As a matter of fact the Federal Council, after consultation with leaders in the various Branches of medical practice, conveyed to the Minister for Health a lengthy list of suggested additions and omissions from the formulary. It may interest Dr. Dark to know that no attention was paid to the suggestions of the Federal Council and the formulary as issued under the Regulations to the *Pharmaceutical Benefits Act*, 1947, is practically identical with the 1944/45 formulary.

Dr. Dark's statement that he cannot remember that the Federal Council had ever objected to formularies such as are used in hospital and lodge practice is also misleading and shows that he has completely failed to appreciate the reason for the Council's objection to the Commonwealth Pharmaceutical Formulary. Dr. Dark knows quite well, or he would if he had read the reports of meetings of the Federal Council that have appeared in THE MEDICAL JOURNAL OF AUSTRALIA, that members of the Federal Council, like other members of the profession, do not object to the use of formularies. Indeed the Council has informed the Minister for Health on several occasions that it would advise the profession to use the Commonwealth Pharmaceutical Formulary wherever appropriate if the Government would accept the prescription of a doctor written on his own prescription form as entitling a patient to free pharmaceutical benefits. With regard to prescribing for lodge patients Dr. Dark knows quite well that the lodge medical officer can prescribe for his patient any drug or preparation in the British Pharmacopœia (excepting sera, vaccines and biological products) and any combination of such drugs or preparations, and that the lodge formulary is simply an aid to prescribing.

Yours, etc.,
J. G. HUNTER,
General Secretary, Federal Council of the British Medical Association in Australia.

135, Macquarie Street,
Sydney,
May 18, 1948.

CANCER "CURES".

SIR: In these days of deluded hopes and disputation bordering on the above subject, it seemed to me that the following prescription that I happened on when cleaning out an outbuilding recently might be of interest, especially as it embraces the escharotic features that are alleged to underlie a reputed cure of recent vintage. Prompted either by the spirit permeating the donor or as a slave to the Hippocratic tradition—and so precluded from putting the "cure" to commercial advantage—I pass on this nostrum for cancer to those members of the profession who should see fit to avail themselves of it.

White oak bark, 4 ozs., bruise well; add urine to cover it. Infuse for four days, boil it until it becomes thick as Molasses. Add 2 ozs. honey and 2 ozs. of strained turpentine gum. To make this plaster Caustic add 2 ozs. of white Vitriol. Spread on soft leather or linen.

Dear Doctor,

As promised to send the above. I could not find it for a long time. It was printed on a book. I am giving you an exact copy. I could not remove the original without destroying it.

August, 14, 1924.

It is perhaps a matter for speculation as to what function the urine fulfils. If we indulge the unworthy assumption that it is there to confuse, confound and mystify and invite a superstitious reverence in a credulous patient, we may incur the cynical suggestion that after all a like circumstance arises in the case of the illegible writing and mysterious signs of much of our prescription writing.

Yours, etc.,
W. J. MCCRISTAL.

149, Macquarie Street,
Sydney,
April 13, 1948.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Medical Statistics.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Dr. H. O. Lancaster will conduct a seminar on "Statistical Aspects of the Differential White Cell Count, the Arneth Count and Derived Indices" on Wednesday, June 9, 1948, at 5.45 p.m., at the School of Public Health and Tropical Medicine, University Grounds.

On Thursday, June 17, at 7.30 p.m., Dr. H. O. Lancaster will give an address to the Statistical Society of New South Wales at History House, 8, Young Street, Sydney, entitled "John Graunt and his Observations on the Bills of Mortality of the City of London (1662)". Those interested will be welcome.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act, 1938-1939*, of New South Wales, as duly qualified medical practitioners:

Anderson, Charlotte Morrison, M.B., B.S., 1945 (Univ. Melbourne), Royal Hospital for Women, Paddington.

Blakely, Eric Robert, M.B., Ch.B., 1944 (Univ. New Zealand), St. Vincent's Hospital, Darlinghurst.

Francis, Harold Hugh, M.B., Ch.B., 1944 (Univ. New Zealand), Women's Hospital, Crown Street, Sydney.

Pedersen, Neils, M.B., Ch.B., 1937 (Univ. Edinburgh), M.R.C.P., 1947 (Edinburgh), 345, Pennant Hills Road, Pennant Hills.

Velvin, Ernest John, M.B., Ch.B., 1941 (Univ. New Zealand), Eye Department, Sydney Hospital, Sydney.

Whitby, Henry, L.M.S.S.A., 1930 (London), c.o. Goyder and Son, 11c, Castlereagh Street, Sydney.

Aarons, Morris Vincent, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Parramatta.

Allison, Robert William, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Marrickville.

Backhouse, Juliet Nancy, M.B., B.S., 1947 (Univ. Sydney), Rachel Forster Hospital, Redfern.

Baker, Cecil Horace, M.B., B.S., 1947 (Univ. Sydney), Hornsby and District Hospital, Hornsby.

Bialoguski, Michael, M.B., 1947 (Univ. Sydney), 226, Sydney Road, Balgowlah.

Boden, Betty, M.B., B.S., 1947 (Univ. Sydney), Eastern Suburbs Hospital, Waverley.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Cary, Wilfred Hazlet, M.B., B.S., 1946 (Univ. Sydney), Royal Hobart Hospital, Hobart.

Langley, Russell Alfred, M.B., B.S., 1946 (Univ. Melbourne), Launceston.

Emery, Warren Frederick, M.B., B.S., 1946 (Univ. Adelaide), Launceston.

Rofe, Owen Frederick, M.B., B.S., 1947 (Univ. Sydney), Royal Hobart Hospital, Hobart.

Wolfers, Philip, M.B., B.S., 1947 (Univ. Sydney), Royal Hobart Hospital, Hobart.

Edwards, Cecil Albert Thomas, M.B., B.S., 1946 (Univ. Melbourne), Launceston.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Vanstone, Stephen Norman, M.B., B.S., 1939 (Univ. Sydney), Prince Henry Hospital, Little Bay.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Caust, David Jannings, M.B., B.S., 1946 (Univ. Adelaide), 115, Watson Avenue, Toorak Gardens.

Kenihan, Robert Austin, M.B., B.S., 1946 (Univ. Adelaide), 200, Goodwood Road, Goodwood.

The undermentioned have been elected members of the South Australian Branch of the British Medical Association:

Wallman, Ian Stuart, M.B., B.S., 1947 (Univ. Adelaide), Mareeba Hospital, Woodville.

Kirkman, David Norton, M.B., B.S., 1947 (Univ. Adelaide), Maitland.

Notice.

AUSTRALIAN RED CROSS SOCIETY.

THE following papers will be delivered at the scientific conference to be held in conjunction with the National Blood Transfusion Committee meetings at the Medical School, Herston, Brisbane, on June 8, 1948, at 8 p.m. (i) Paper by R. T. Simmons entitled "The Organization of Blood Transfusion Services in England and Methods of Rh Testing". (ii) Paper by Dr. R. Jakobowicz entitled "Some Observations on Anti-O Agglutinins". (iii) Paper by Dr. R. V. Sellwood entitled "New Approaches to the Basic Physiology of Circulation with Particular Reference to Blood Transfusion". (iv) Short report by Dr. J. O'Reilly on "The Incidence of Homologous Serum Jaundice in New South Wales".

The National Council of the Australian Red Cross Society has issued an invitation to members of the British Medical Association in Brisbane and to Fellows and Members of the Royal Australasian College of Physicians who are visiting Brisbane in connexion with the meetings of the College to attend the scientific meeting and take part in the discussion.

Medical Appointments.

Dr. E. F. West has been appointed a member of the Physiotherapists Board of South Australia.

Dr. H. McI. Birch, Superintendent of Mental Institutions, has been appointed Deputy Director-General of Medical Services, South Australia.

Dr. H. W. Linn has been appointed medical superintendent, Northfield Wards, on the temporary staff of the Hospitals Department, South Australia.

Dr. C. W. England has been appointed government medical officer, Department of Public Health, Newcastle, New South Wales.

Dr. B. L. W. Clarke has been appointed a member of the Queensland Radium Institute, in pursuance of the provisions of *The Health Acts, 1937 to 1946*, of Queensland.

Dr. H. Crawford, Dr. P. Mitchell and Dr. P. R. Patrick have been appointed members of the National Fitness Council of Queensland.

Books Received.

"Modern Psychiatry in Practice", by W. Lindesay Neustatter, M.D., B.Sc., M.R.C.P.; Second Edition; 1948. London: J. and A. Churchill, Limited. 8" x 5¼", pp. 284. Price: 12s. 6d.

"Diseases of the Eye", by Sir John Herbert Parsons, C.B.E., D.Sc., F.R.C.S., F.R.S., and Sir Stewart Duke-Elder, K.C.V.O., M.A., D.Sc., Ph.D., M.D., F.R.C.S., Hon.DSc. (North Western); Eleventh Edition; 1948. London: J. and A. Churchill, Limited. 8" x 5¼", pp. 740, with many illustrations, some coloured. Price: 30s.

"Clinical Ophthalmology: For General Practitioners and Students", by H. M. Traquair, M.D., F.R.C.S., Ed.; 1948. London: Henry Kimpton. 8½" x 5½", pp. 286, with illustrations, some coloured. Price: 25s.

"Tuberculosis in the British Zone of Germany with a Section on Berlin", report of an inquiry made in September-October, 1947, by M. Daniels, M.D., D.P.H., and P. D'Arcy Hart, M.D., F.R.C.P.; 1948. London: His Majesty's Stationery Office. 9¼" x 6", pp. 32. Price: 6d.

"High-Ho to London: Incidents and Interviews on a Leisurely Journey by Air from Australia to Britain" with Frank Clune; 1948. Sydney, London: Angus and Robertson. 8¼" x 5¼", pp. 276, with illustrations. Price: 12s. 6d.

Diary for the Month.

JUNE 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 2.—Victorian Branch, B.M.A.: Branch Meeting.
JUNE 2.—Western Australian Branch, B.M.A.: Council Meeting.
JUNE 3.—South Australian Branch, B.M.A.: Council Meeting.
JUNE 3.—New South Wales Branch, B.M.A.: Special Groups Committee.
JUNE 4.—Queensland Branch, B.M.A.: Branch Meeting—Bancroft Oration.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch **(Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.**

Victorian Branch **(Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.**

Queensland Branch **(Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health) Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.**

South Australian Branch **(Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.**

Western Australian Branch **(Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.**

Editorial Notices.

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